DISSERTATION TITLED

"BETAINE THERAPY AND ITS INFLUENCE ON OUTCOME OF ACUTE ON CHRONIC ALCOHOLIC LIVER DISEASE"

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CERTIFICATE

This is to certify that the dissertation "**BETAINE THERAPY AND ITS INFLUENCE ON OUTCOME OF ACUTE ON CHRONIC ALCOHOLIC LIVER DISEASE"** is a bonafide work done by **Dr.VENKATAKRISHNAN R.** Post Graduate Student, Institute of Internal Medicine, Madras Medical College, Chennai-3, during March 2015 to August 2015 in partial fulfillment of the University Rules and Regulations for the award of MD Branch – I General Medicine, under our guidance and supervision, during the academic year 2013 - 2016.

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DECLARATION

I solemnly declare that the dissertation entitled "**BETAINE THERAPY AND ITS INFLUENCE ON OUTCOME OF ACUTE ON CHRONIC ALCOHOLIC LIVER DISEASE**" is done by me at Madras Medical College, Chennai-3 during March 2015 to August 2015 under the guidance and supervision of **Prof.K.S.CHENTHIL, M.D.,** to be submitted to The Tamilnadu Dr. M.G.R Medical University towards the partial fulfillment of requirements for the award of M.D. DEGREE IN GENERAL MEDICINE BRANCH-I.

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CONTENTS

ABBREVIATIONS

Ampk	-	AMP-activated protein kinase
ACC	-	Acetuly coA carboxylase
ALD	-	Alcoholic liver disease
HCC	-	Hepatocellular carcinoma
PNPLA3	-	Patatin-like phospholipase domain-containing protein 3
HIF	-	Hypoxia Inducible Factor
iNOS	-	Inducible Nitric Oxide Synthase
TNF	-	Tumour Necrosis Factor
LPS	-	Lipopolysaccharide
TLR	-	Toll Like Receptor
IL	-	Interleukin
STAT	-	Signal Transducer and Activator of Transcription
TMG	-	Trimethyl Glycine
Nafld	-	Non Alcoholic Fatty Liver Disease
CNS	-	Central nervous system
IFN	-	Interferon
TGF	-	Transforming Growth Factor
OLT	-	Orthotopic Liver Transplantation

DF	-	Discriminant Factor
MELD	-	Model for End Stage Liver Disease
ROS	-	Reactive Oxygen Species
NK	-	Natural Killer
NF-kB	-	Nuclear Factor –kB
EtOH	-	Ethanol
ER	-	Endoplasmic Reticulum
PKC	-	Protein Kinase C
PPAR	-	Peroxisome proliferator activated Receptor
NADH	-	Nicotinamide Adenine Dinucleotide Hydrogen
TRIF/RIF	-	TIR-domain-containing adapter-inducing interferon-β
HSC	-	Haematopoetic stem cell

INTRODUCTION

INTRODUCTION

With increasing consumption of alcohol and increasing burden of chronic liver disease patients in India and across the world, research efforts have only managed to raise more questions than provide answers to the demands of both logic, science and clinicosocial burden of patients with this disease. With more definitive and curative treatments becoming available for liver diseases due to viral etiologies and intermediary metabolites, it is alcoholic liver disease that seems to have been orphaned with respect to finding cures and clear potential targets in the pathological process. Counterintuitive to that which may be believed, perhaps more breakthroughs are achieved by finding cures by trial and error and then subsequently managing to elucidate the exact reason for its curative potential, which may thus give rise to a subsequent breakthrough in the pathophysiology of the inciting process. Betaine is a drug that acts as a methyl group donor whereby it reduces the fatty burden and interrupts the pathogenic process in non alcoholic fatty liver disease. Its use has yet to be explored on alcoholic liver disease. It must also be impressed that it is in no way a random choice of drug as multiple trials have been done in the past but with other diseases and this particular scenario has perhaps not been satisfactorily subjected to the potential of betaine. No evidence has emerged theoretically precluding the utility of this drug in the above scenario. It is with this inquisitiveness that this possibility is being explored.

AIMS

AND

OBJECTIVES

AIMS AND OBJECTIVES

- To study the “BETAINE THERAPY AND ITS INFLUENCE ON OUTCOME OF ACUTE ON CHRONIC ALCOHOLIC LIVER DISEASE’’

REVIEW

OF

LITERATURE

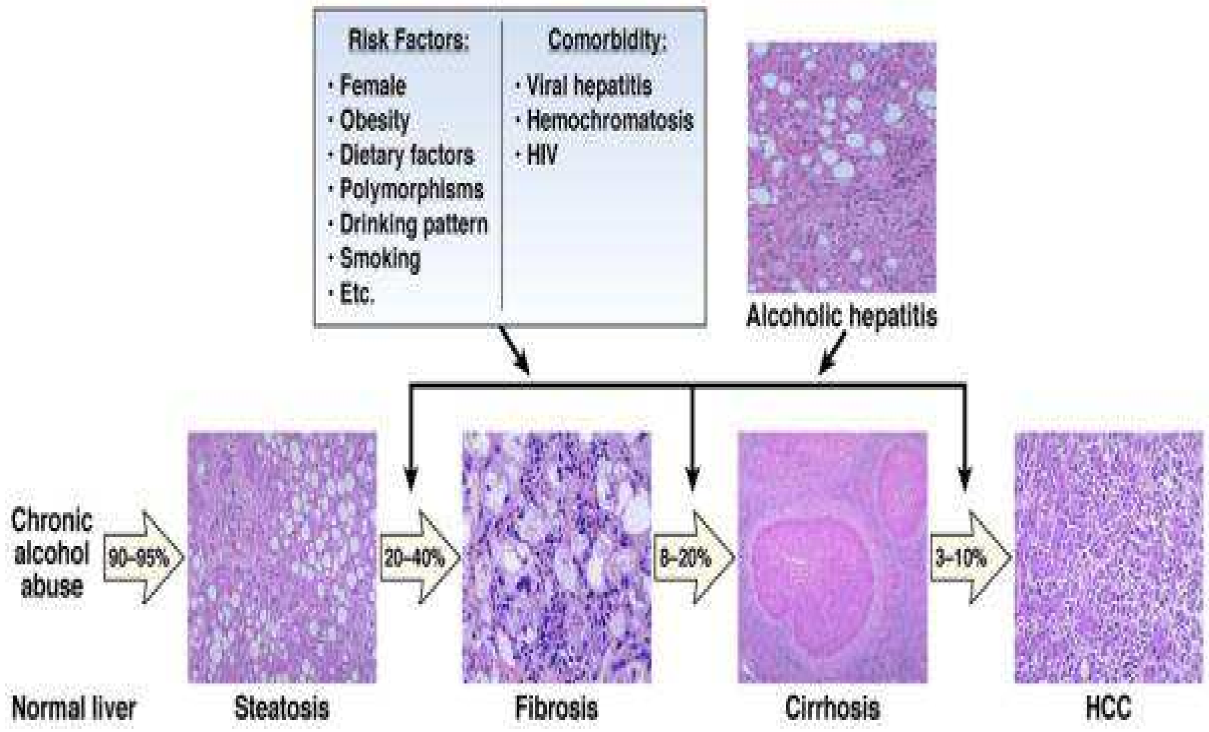
ALCOHOLIC LIVER DISEASE

Alcoholic liver disease (ALD) is a major cause of chronic liver disease worldwide and can lead to fibrosis and cirrhosis. Forty eight percent of all cirrhosis related deaths are alcohol related (2011). The spectrum of disease ranges from fatty liver, alcoholic hepatitis which leads to fibrosis, cirrhosis, and finally to hepatocellular carcinoma

RISK FACTORS

Younger age of initiation of the alcohol habit has shown to be a risk factor for development of alcoholic liver disease. Female sex is a strong risk factor due to increased ratio of body fat and high oestrogen levels. Obesity is a separate risk factor which escalates fibrosis progression and the development of cirrhosis in ALD. Experiments show that the synergistic effects of obesity and alcohol abuse involve the smooth endoplasmic reticulum response to cell stress, stimulation or macrophages and end organ resistance to adiponectin. Daily Drinking as compared to binge drinking or sporadic drinking is associated with a higher incidence of liver disease. Smoking is an independent risk factor. Genetic factors also play a role especially PNPLA3 mutations. Variations in patatin-like phospholipase domain-containing protein 3 (*PNPLA3*) are associated with liver disease in whites.Reduced amount of alcohol dehydrogenase enzyme in the stomach mostly seen in female patients is an important risk factor.

FIGURE 1



Prolonged alcohol drinking has synergistic effects with Hepatitis virus B or C and HIV infection, nonalcoholic fatty liver disease, and hemochromatosis to accelerate progression of liver diseases. For example, many patients with viral hepatitis consume alcohol, which escalates progression of liver disease.

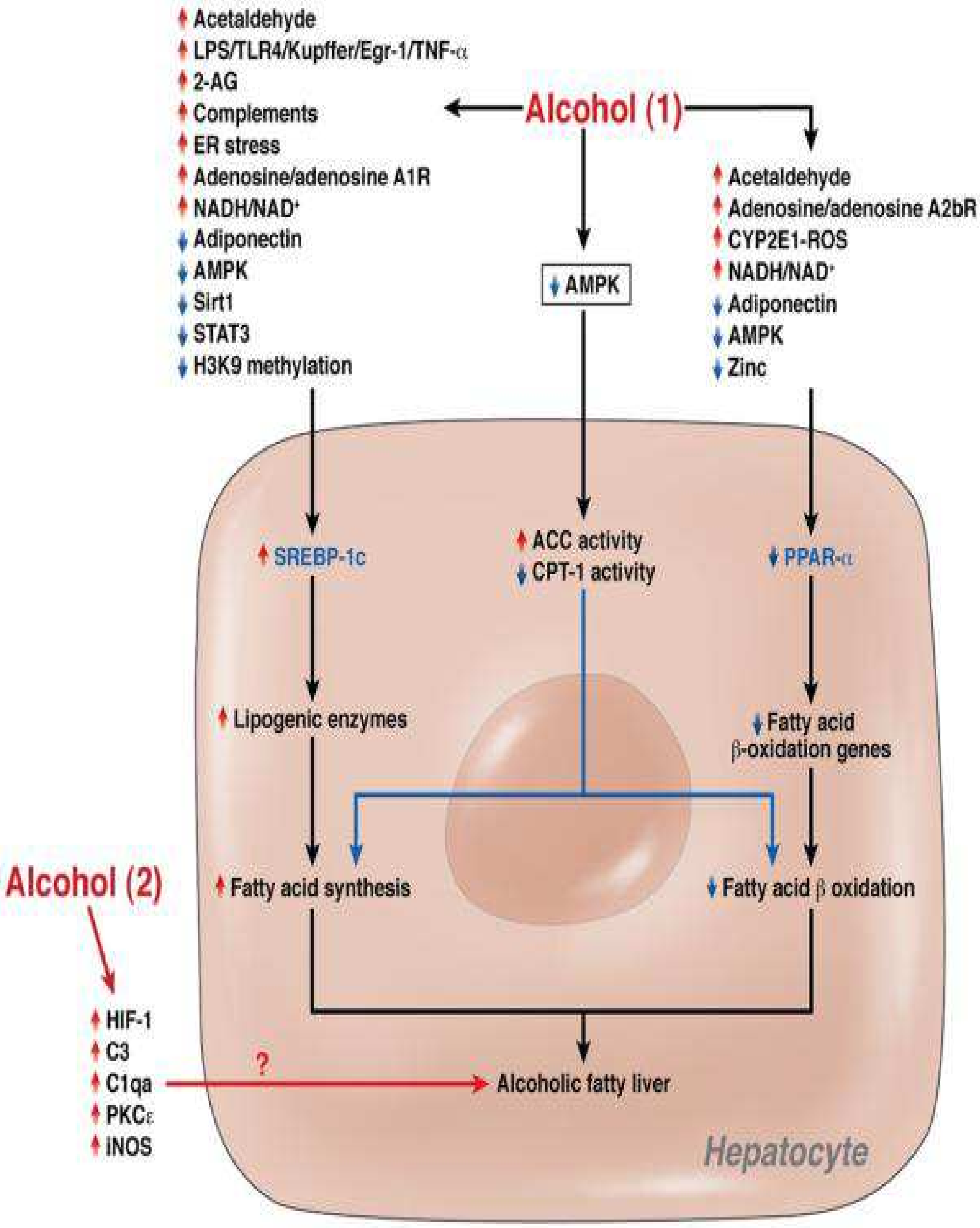
PATHOGENESIS

Steatosis: Fatty change, the preliminary reaction of the liver to alcohol abuse, is typified by the increased presence of lipids (mainly triglycerides, phospholipids, and cholesteryl esters) in liver cells. Preliminary studies suggested that ethanol intake increases the proportion of reduced nicotinamide adenine dinucleotide/oxidized nicotinamide adenine di-nucleotide in liver cells, which inhibits mitochondrial β-oxidation of fatty acids and results in fatty change. Ethanol consumption has also been found to enhance the transport of fats to the liverliver from the small intestine, increasing mobilization of fatty acids from adipose tissue and uptake of fatty acids by the liver. However, the contribution of these mechanisms to the development of steatosis after long-term alcohol consumption is not clear and requires further investigation.

Liver from the gut, enhancing movement of fatty acids from adipose tissue. However, the importance of these events in the context of fatty change after chronic ethanol intake is uncertain and mandates subsequent research.

Recent studies show that alcohol exposure, alters lipid metabolism-associated transcription factors. This stimulates lipogenesis and inhibits fatty acid oxidation. Thus alcohol increases fatty acid synthesis in hepatocytes via up-regulation of sterol regulatory element-binding protein 1c (SREBP-1c), a molecule that stimulates fatty acid synthesis via up-regulation of lipid forming genes. Alcohol consumption increases transcription of SREBP-1c gene via its metabolite acetaldehyde directly or by activating pathways and molecules that up-regulate its expression, such as the endoplasmic reticular stress reaction and Toll like receptor 4. Alcohol may also decrease factors that reduce SREBP-1c expression, such as AMP-activated protein kinase (AMPK), and adiponectin. Blockade of *SREBP-1c* in animal models reduced alcohol related fatty change.

FIGURE 2



Alcohol intake decreases fatty acid oxidation in liver parenchyma through inactivation of the peroxisome proliferator-activated receptor (PPAR)-α, a nuclear hormone receptor. Ethanaldehyde, but not ethyl alcohol by itself, directly inhibits the functionality of PPAR-α in liver cells.

Alcohol intake can also indirectly inhibit PPAR-α via up-regulation of cytochrome P2E1-derived free radical stress and adenosine, thereby inhibiting PPAR-α, or reducing zinc and adiponectin, which stimulate PPAR-α. Alcohol may also cause changes in function of enzymes involved in fat metabolism by inhibiting AMPK, which reduces fat metabolism and fatty liver. AMPK acts as a serine-threonine kinase that inactivates acetyl-CoA carboxylase (ACC). Being a rate limiting step of fatty acid synthesis, its inactivation is crucial. This also results in reduced levels of malonyl-CoA, a reactant in the fatty acid synthesis cascade which blocks carnitine palmitoyltransferase 1, a rate-limiting step in the fatty acid oxidation pathway.

AMPK through its phosphorylating activity inhibits SREBP activity in liver cells, decreasing fatty change. Thus AMPK would normally inhibit lipogenesis and up-regulate fatty acid oxidation by inactivating ACCEthanol intake blunts AMPK activity in the hepatocytes, causing unchecked functioning of ACC and carnitine palmitoyltransferase 1, all of which have a crucial role in ALD. Alcohol related fatty change is markedly diminished in many strains of knockout mice with including HIF-1 and iNOS enlightening us of the possible gene interactions which contributes to the pathogenesis of alcoholic fatty liver. The possible implications to research outcomes are exhilerating.

Phagocytosis plays an undeniable role in scavenging out fat globules in liver cells. Chronic ethanol intake blunts this autophagy response. Although short-term alcohol intake stimulates phagocytosis by creating free radical species and blocking the mammalian target of rapamycin, suggesting that acute alcohol intake could have a balancing act that helps to adjust and halt the development of fatty change during the initial insults of alcohol.

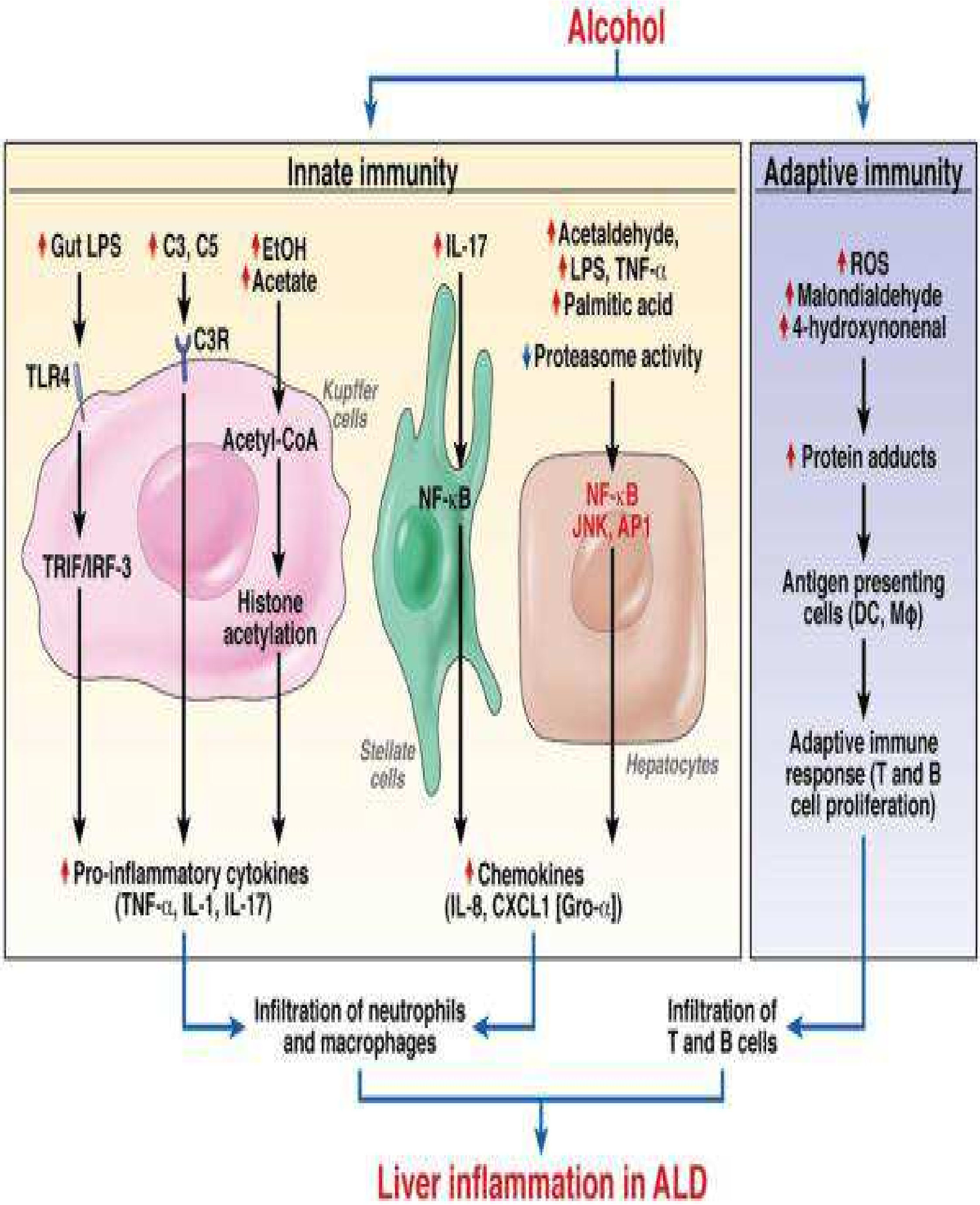
Alcoholic Hepatitis: Alcoholic hepatitis is a syndrome described by affection and inflammation of the liver by inflammatory cells leading to hepatocellular injury. AH develops in those affected with pre-existing fatty change and usually goes on to progressive fibrosis. The prevalence of alcoholic hepatitis is not clearly known. Alcoholic hepatitis can range from mild injury to severe, life-threatening injury. The histologic characteristics of AH include steatosis, centrilobular ballooning of hepatocytes, presence of polymorphs, Mallory hyaline inclusions, and a background of ominous chicken wire–like fibrotic deposition. Many patients, there may actually be underlying cirrhosis.A large body of evidence indicates that many factors contribute to alcohol-induced inflammation

Toxicity of alcohol: In liver cells, ethanol is metabolized into acetaldehyde by alcohol dehydrogenase in the cytoplasm, cytochrome P-450 in microsomes, (and in perixosomes through catalase). This creates free radical species which results in lipid peroxidation, depletion of glutathione, and *S*-adenosylmethionine insufficiency; which subsequently result in hepatocytes to stress and injury. Acetaldehyde is then rapidly metabolized into acetic acid-acetate by aldehyde dehydrogenase. Acetaldehyde is a reactive compound and it is highly toxic to liver cells because it forms dangerous protein changes and DNA adducts that promote lipid peroxidation, glutathione depletion, and mitochondrial damage. This acetate is immediately released from the liver into the blood stream and is then metabolized into carbon dioxide in the citric acid cycle in other organs having a rich mitochondrial content like skeletal and cardiac muscles. Acetate admittedly has little direct hepatotoxicity, it is important in the inflammation in patients with AH via the resultant stimulation of proinflammatory cytokines in macrophages.

Role of innate immunity: Ethanol intake causes gut dysbiosis and bacterial over-growth but also enhances gut permeability and the translocation of Lipopolysaccharide from bacteria to the liver. These changes explain the increased levels of LPS observed in alcoholic liver disease. In Kupffer cells (liver macrophages), LPS binds with TLR4 to activate the MyD88-independent (TRIF/IRF-3) signaling pathway thus causing increase of free radical stress and inflammatory molecules, like TNF-α, that cause hepatocellular damage. Ethanol intake activates complement C3 and C5, which subsequently activate Kupffer cells by binding to complement receptors on these cells. This complement activation also results In TNF-alpha release. Although activation of TLR4 and complement factors do also cause Kupffer cells to produce cytokines such as interleukin (IL)-6 and the anti-inflammatory cytokine IL-10, these cytokines activate STAT3 in Kupffer cells to protect from alcohol-induced liver injury. Thus, the activation of innate immunity components not only triggers alcoholic liver injury but also triggers protective responses that reduce alcohol-induced hepatocellular damage.

Liver Cell apoptosis: Hepatocyte apoptosis is an increasingly important feature of alcoholic liver disease. Apoptosis results from multiple pathways, including direct hepatotoxicity, free radical injury,, and induction of proapoptotic molecules (TNF-$\alpha$and Fas ligand).

FIGURE - 3



Alcohol
Innate immunity
Adaptive immunity
Gut LPS
C3, C5
EtOH
Acetate
IL-17
Acetaldehyde,
LPS, TNF-α
Palmitic acid
Proteasome activity
TLR4
C3R
Kupffer cells
Acetyl-CoA
TRIF/IRF-3
Histone acetylation
NF-κB
NF-κB
JNK, AP1
Stellate cells
Hepatocytes
Pro-inflammatory cytokines (TNF-α, IL-1, IL-17)
Chemokines (IL-8, CXCL1 [Gro-α])
ROS
Malondialdehyde
4-hydroxynonenal
Protein adducts
Antigen presenting cells (DC, Mϕ)
Adaptive immune response (T and B cell proliferation)
Infiltration of neutrophils and macrophages
Infiltration of T and B cells
Liver inflammation in ALD

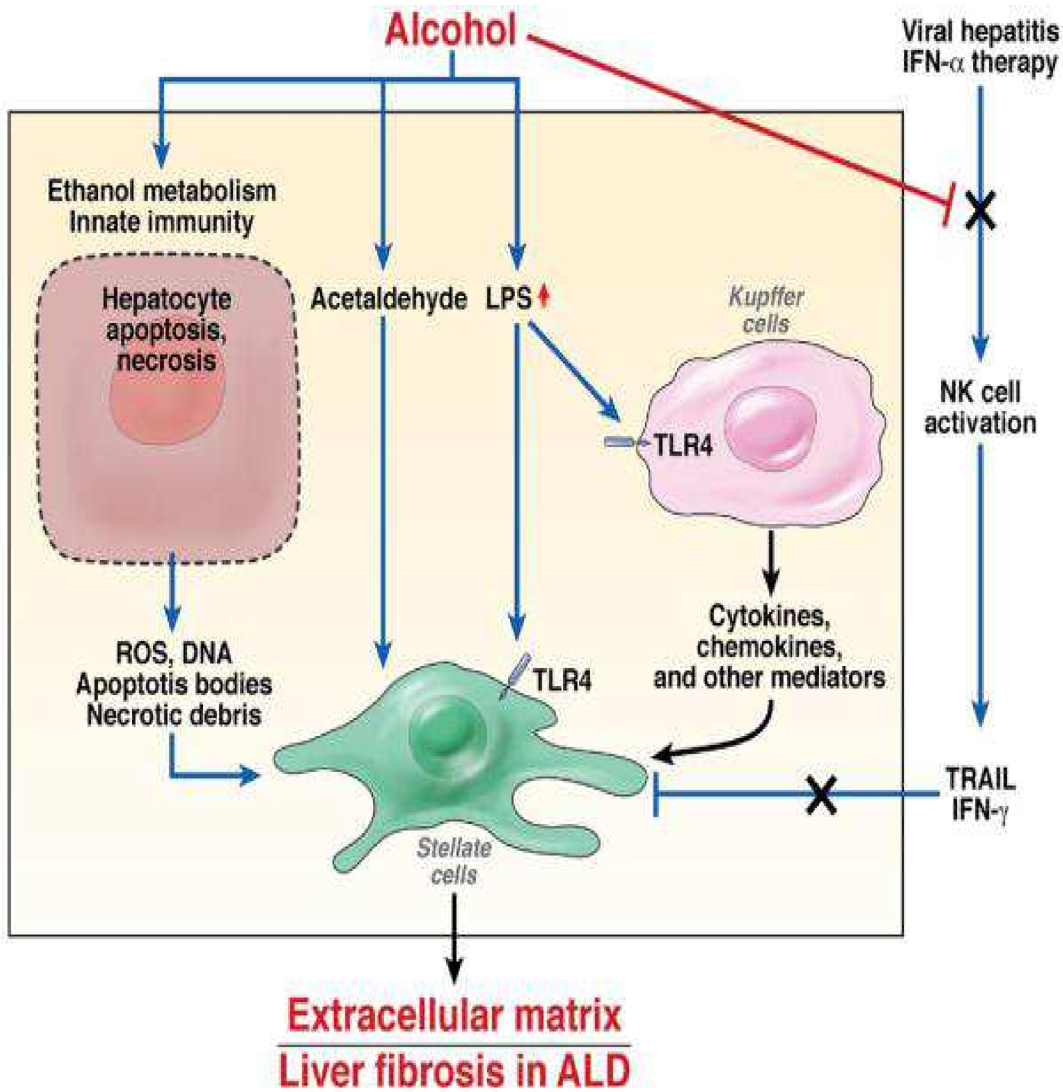
Role of polymorphs: presence of polymorphs in the liver parenchyma is a stellar feature of AH. Up-regulation of pro-neutrophilic cytokines like IL-8, and IL-17 in liver causes this infiltration. Many factors stimulate cells to produce these cytokines in patients with AH. These include LPS, acetaldehyde, TNF-α,, and blunting of proteasome activities. IL-17, which found in high levels in patients with AH, causing polymorph attraction but also stimulates sinusoidal stellate cells to produce IL-8 and CXCL1; which results in infiltration of the parenchyma with polymorphs. Also, IL-8 and CXCL1 are secreted by activated stellate cells and Kupffer cells, which promote further neutrophil recruitment. Many other chemokines and cytokines are markedly up-regulated promoting infiltration of neutrophils during progression of AH. Neutrophil infiltration strongly correlates with the severity of AH, but there is no direct evidence in animal experiments to ascertain the role of neutrophils in pathogenesis

Role played by adaptive immunity: Individuals with alcoholic hepatitis have enhanced amounts of antibodies against lipid peroxidation products and there is a high amount of T cells in the liver, suggesting that of adaptive immunity plays a role in alcoholic liver disease, including alcoholic hepatitis. Chronic ethanol intake increases free radical stress, which generates lipid peroxidation products such as malondialdehyde and 4-hydroxynonenal; which can modify many biomolecules, inducing the formation of protein products which can behave like epitopes to activate the corresponding adaptive response. However, the exact ways by which adaptive immune responses produce liver cell toxicity and organ dysfunction in subjects with alcoholic hepatitis are under study.

Blockade of regenerative response **:** The liver is a resilient organ capable of great recovery; regeneration of liver cells occurs principally by reproduction by remaining unaffected liver cells, cholangioepitheliocytes, and vessels wall structures. In conditions where these cells are prevented from reproducing, native liver progenitor cells, ductular hepatocytes, increase in number and transform into liver cells or epithelial cells of the biliary tract. There has been a study which showed reduced liver cell division post hepatectomy in chronic ethanol intake but the direct morbidity ethanol causes on liver cell reproduction and proliferation is yet to be understood. It is not farfetched to now imagine that chronic ethanol intake not only causes liver cell apoptosis but significantly hinders liver cell regeneration, adding to another of the multivarious ways alcohol may induce hepatocytic damage in alcoholic liver disease

Collagenic regeneration of liver: Liver fibrosis is a regenerative response to practically all forms of chronic liver injury; it is typified by predominant deposition of extracellular matrix proteins especially collagen. The major source of this collagen and other extracellular matrix proteins is by haematopoetic stem cells. Other cells involved in this are portal fibroblasts and myofibroblasts derived from the haematopoetic marrow. The haematopoetic stem cells are derived from autocoids, angiogenic factors, cytokines, chemokines, chemicals of innate immunity, and other molecules which are secreted due to liver cell injury. Fibrogenic response, being a typical terminal common pathway for prolonged and persistent significant liver cell injury, is similar in alcohol related injury as is with other causes of liver injury, although some aspects of the fibrogenic response cascade may be unique to alcohol.

FIGURE - 4

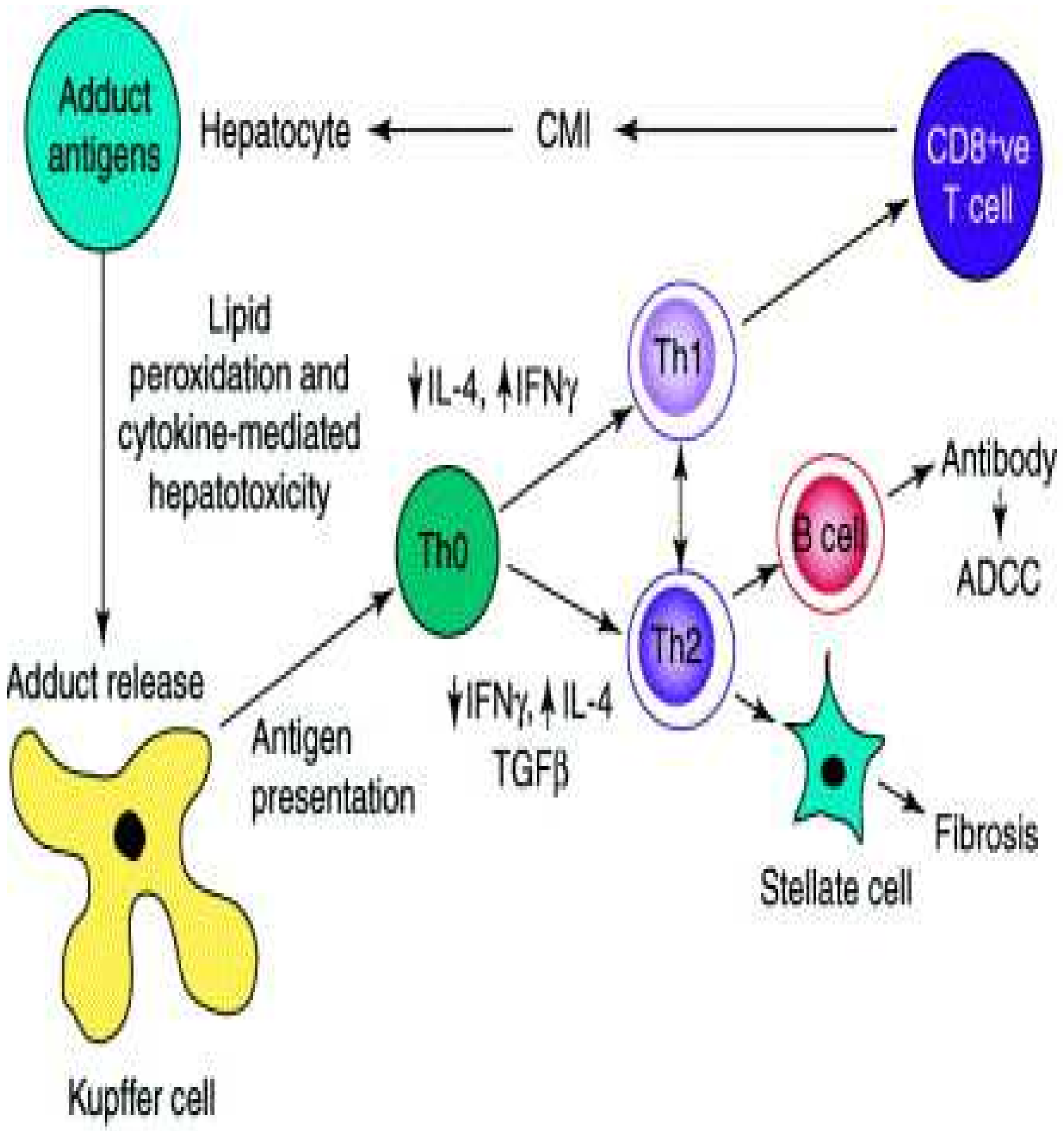


LPS activates TLR4: Most patients with alcoholic liver disease have increased levels of Lipopolysaccharide. LPS can directly or indirectly stimulate the haematopoetic stem cells. First, it can directly activate them via Toll like Receptor-4. And secondly, it can activate free radical stress in the Kupffer cells and a cytokine increase which in turn activate the stem cells. Angiogenesis, which can also result in fibrogenesis, is activated by TLR-4 mediated stimulation of sinusoidal endothelial cells. Thus one can safely say that TLR-4 signaling plays a major role in the fibrotic process. There was a recent report using chimeric mice in which wild-type mice underwent transplant with bone marrow cells from TLR4 knockout mice and vice versa, which showed inhibition or slow down of the fibrotic process when TLR-4 mediated mechanisms were shut down in the host.

Activation of HSCs by acetaldehyde: Acetaldehyde is generated mainly by liver cells and acts on HSCs in a paracrine fashion; it linearly increases synthesis of collagen I in HSCs via amplification of various signaling cascades and regulatory factors. Acetaldehyde also combines swiftly with cellular components, producing dangerous adducts such as malondialdehyde, 4-hydroxynonenal, and malondialdehyde-acetaldehyde, which result in a sustained HSC activation.

Alcohol inhibiting antifibrotic mechanisms**:** A major anti fibrotic mechanism in cells is in the form of activated natural killer cells which destroy haematopoeticstem cells or by producing interferon-γ,which induces cell cycle checks in the stem cells. Long term alcohol consumption results in weakening and stunting of this response. This could potentially explain fibrosis in alcohol intake as well as enhanced premature fibrosis in patients with viral hepatitis who also consume alcohol.

FIGURE - 5



Adduct antigens
Hepatocyte
CMI
CD8+ve T cell
Lipid peroxidation and cytokine-mediated hepatotoxicity
↓IL-4, ↑IFNγ
Th1
Th0
Antibody
ADCC
B cell
Th2
Adduct release
Antigen presentation
↓IFNγ, ↑IL-4 TGFβ
Fibrosis
Stellate cell
Kupffer cell

TREATMENT

People who are suffering from this terrible disease must be subjected to treatments to kick the habit, i.e. deterrents. It is an established fact that continued alcohol intake is the single most important risk factor for further disease progression. One must be able to recognize and accept the problem first.

CAGE Questionnaire and AUDIT questionairre:

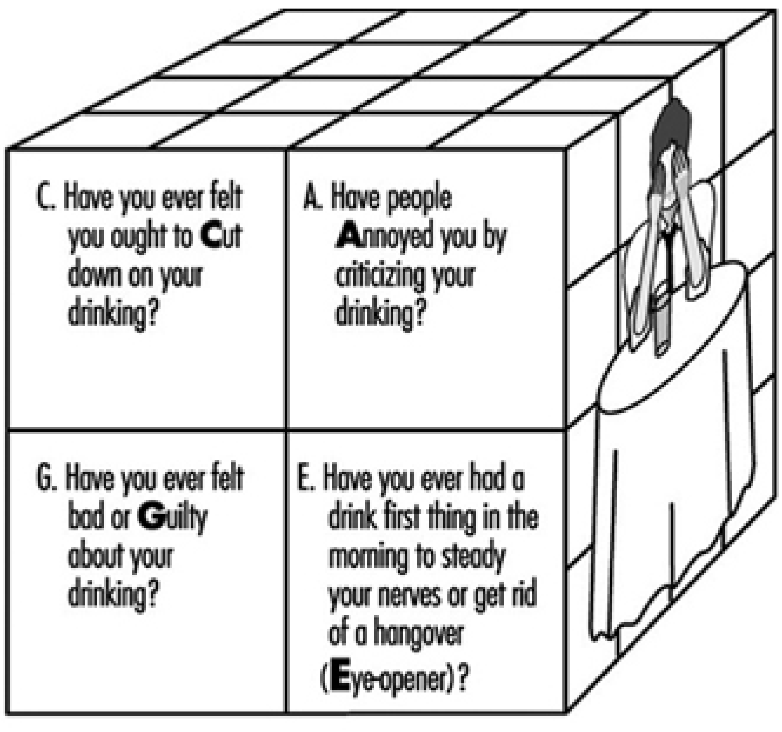


FIGURE - 6

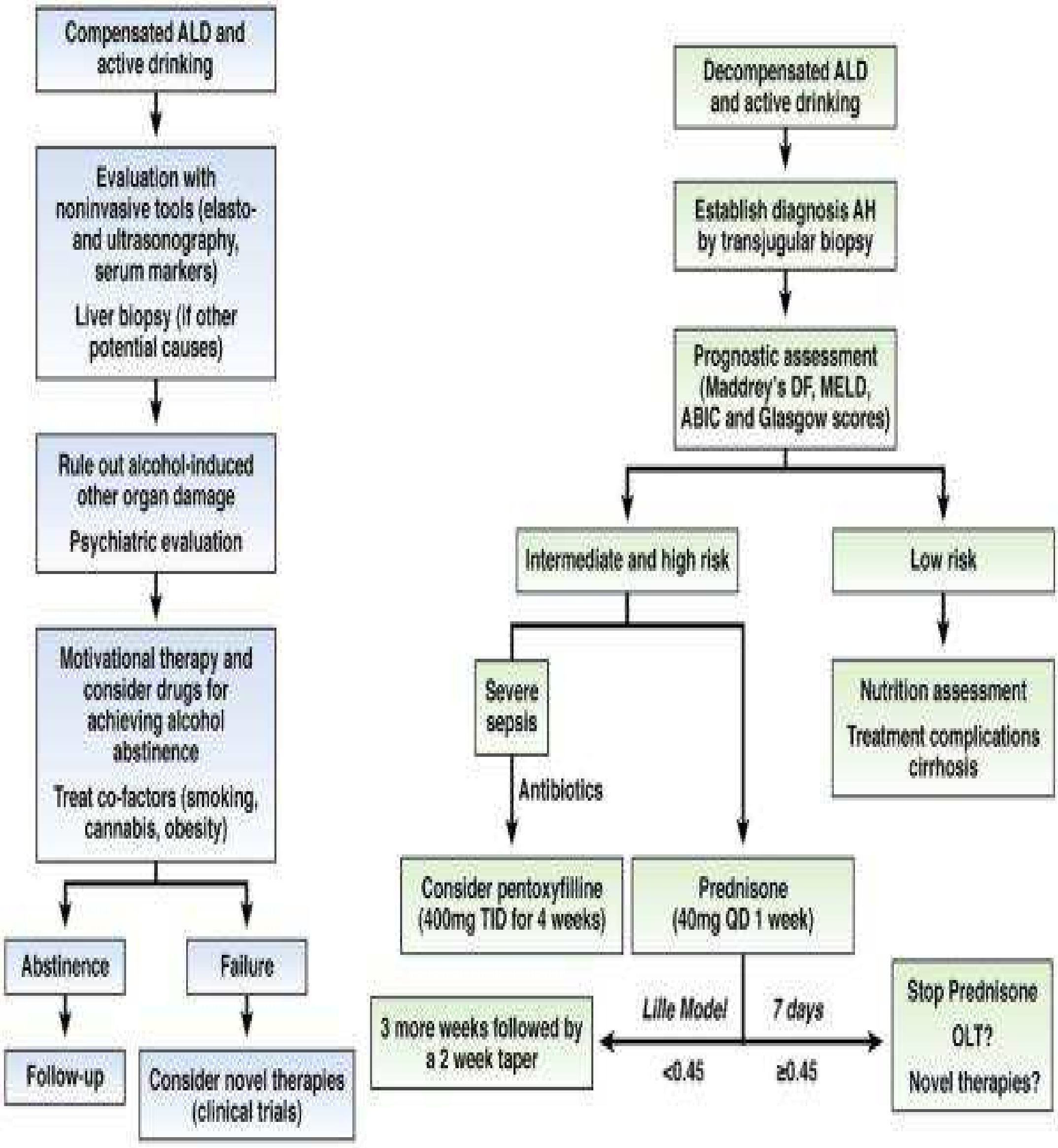
The Alcohol Use Disorders Identification Test (AUDIT)		
1	How often do you have a drink containing alcohol?	Never / Monthly or less / 2-4 times a month / 2-3 times a week / 4 or more times a week
2	How many standard drinks containing alcohol do you have on a typical day when drinking?	1 or 2 / 3 or 4 / 5 or 6 / 7 to 9 / 10 or more
3	How often do you have six or more drinks on one occasion?	Never / Less than monthly / Monthly / Weekly / Daily or almost daily
4	During the past year, how often have you found that you were not able to stop drinking once you had started?	Never / Less than monthly / Monthly / Weekly / Daily or almost daily
5	During the past year, how often have you failed to do what was normally expected of you because of drinking?	Never / Less than monthly /Monthly / Weekly / Daily or almost daily
6	During the past year, how often have you needed a drink in the morning to get yourself going after a heavy drinking session?	Never / Less than monthly / Monthly / Weekly / Daily or almost daily
7	During the past year, how often have you had a feeling of guilt or remorse after drinking?	Never / Less than monthly / Monthly / Weekly / Daily or almost daily
8	During the past year, have you been unable to remember what happened the night before because you had been drinking?	Never / Less than monthly / Monthly / Weekly / Daily or almost daily
9	Have you or someone else been injured as a result of your drinking?	No / Yes, but not in the past year / Yes, during the past year
10	Has a relative or friend, doctor, or other health worker been concerned about your drinking or suggested you cut down?	No / Yes, but not in the past year / Yes, during the past year

Another factor significant in this scenario is that of transplantation and the fact that patients who continually expose themselves to the toxic effects of alcohol, preclude themselves from eligibility for transplantation. Patients need to be taken care of under special rehabilitation programs. There are many possible options for pharmacological treatment for alcohol dependence. The irreversible inhibitor of alcohol dehydrogenase, Disulfiram, is often used, but its use must be weighed against the risk of serious hepatotoxicity in patients who already have compromised liver function and reserve. Another drug, Acamprosate is also similar in side effect profile. The GABA-B agonist, Baclofen is also useful and may, in fact, be the safest of the three fr aiding abstinence. The opioid antagonist naltrexone has been found to decrease relapse, although its efficacy is limited.

Currently there are no approved antifibrotic drugs to prevent disease progression in patients with alcoholic liver disease. There do exist only ways to assess the extent of fibrosis using imagine or biopsy studies. There are many confounding factors that need to be taken in during trials like the positive outcome that may arise due the fact that patients alter drinking patterns once they are fully enrolled in trials.

When it comes to full blown cirrhosis, patients who have a reduced drinking pattern have a much slower progression of cirrhosis subsequently, than patients who do not. Apropos, patients who drink on top of existing cirrhosis predispose themselves to subsequent steatohepatitis of the existing parenchyma and decompensation. The only established facts about treatment of alcoholic cirrhosis remain that firstly, the regimens are the same as that for cirrhosis for other causes, and secondly, the only addition therapeutic intervention currently of established value, is alcohol abstinence.

Alcoholic hepatitis, represents a potentially serious aspect of alcoholic liver disease; its treatment has been described by the following:


Compensated ALD and active drinking
Evaluation with noninvasive tools (elasto- and ultrasonography, serum markers)
Liver biopsy (if other potential causes)
Rule out alcohol-induced other organ damage
Psychiatric evaluation
Motivational therapy and consider drugs for achieving alcohol abstinence
Treat co-factors (smoking, cannabis, obesity)
Abstinence
Failure
Follow-up
Consider novel therapies (clinical trials)
Decompensated ALD and active drinking
Establish diagnosis AH by transjugular biopsy
Prognostic assessment (Maddrey's DF, MELD, ABIC and Glasgow scores)
Intermediate and high risk
Low risk
Severe sepsis
Antibiotics
Nutrition assessment
Treatment complications cirrhosis
Consider pentoxyfilline (400mg TID for 4 weeks)
Prednisone (40mg QD 1 week)
3 more weeks followed by a 2 week taper
Lille Model
7 days
<0.45
≥0.45
Stop Prednisone
OLT?
Novel therapies?

TREATMENT OF AH

SUPPORTIVE TREATMENT :

Patients with alcoholic hepatitis may be sick enough to require critical care. Depending on the degree of encephalopathy, airway protection may be necessary. During procedures, usage of CNS suppressants like benzodiazepines should not be used. Given that most of these patients may be intoxicated at the time of presentation or may have a severely depleted thiamine store, restoration of thiamine levels will also have to be taken care of, lest Wernicke's encephalopathy is precipitated. Owing to the severe susceptibility to infections these people may be prone for, or the fact that very often it is an infection that precipitates these episodes, antibiotics should be administered either therapeutically or empirically.

CORTICOSTEROIDS:

The addition of steroids to alcoholic hepatitis treatment repertoire was by Maddrey et al in 1978 when a randomized double blinded control trial which studied the use of corticosteroids in the test arm in 55 patients with alcoholic hepatitis. But since then there have been several meta-analyses that have refuted claims of utility of steroids. Also, multiple meta-analyses alleged that steroids enhanced duration of survival amongst subjects , but one in particular alleged bias in some trials, therefore not advocating its use.

These conflicting opinions notwithstanding, the American Association for the Study of Liver Diseases recommends the prescription of corticosteroids in the setting of a severe alcoholic hepatitis, where a Maddrey's discriminant factor is greater than 32, and/ or the patient develops profound hepatic encephalopathy. Consequently, these steroids also worsen risk of infection and any patient with a significant evidence of infection when under steroid therapy must be monitored. Another index exists which suggests the benefit of the use of steroids in such patients at the end of one week of steroid therapy- called the Lille score, a score of >0.45 indicating poor benefit from steroids and a six month survival rate of less than one-fourth of patients. Some laboratory studies have shown that in clinical cases where alcoholic hepatitis is severe, the usual accompanying steroid refractoriness is reduced by Theophylline, a fact that still needs to be established in-vivo. Thus there may be a bigger role for steroids after all.

PENTOXIFYLLINE:

Another agent of particular interest is this phosphodiesterase inhibitor. Its main action seems to be to suppress circulating IFN-alpha levels by reducing its transcription. Historically it has been used to treat those patients who simply cannot be given steroids. The reduced mortality hitherto suggested at for pentoxifylline may be due to its protective effect over hepatorenal syndrome. Recent studies, though, have shown that the effects of this drug must be viewed with some amount of caution as it may not be beneficial at all. The efficacy of this drug may be very much reduced compared to steroids as those who were treated with this drug as a rescue therapy for steroid refractoriness have not improved.

Anti-TNF-α agents :

As TNF- α has been strongly implicated in the pathogenesis in animal models, trials have been conducted where inhibitors of this cytokine have been used. Although there may have been a benefit in terms of improvement of the hepatitis, they greatly increase mortality and morbidity due to the increased risk of infections. Thus currently the use of these agents is not advised.

NUTRITION THERAPY:

Directly due to behaviour and due to malabsorption, lots of these patients experience malnourishment which needs to be addressed. Apart from the overall patient benefit that comes with better nourishment, patients also experience an improvement in survival in terms of better liver function as witnessed by more rapid recovery of liver function tests in the short term and an improved histology profile in those whose nutritional status has been improved.

SAMe:

This is a methyl donor which has been found in many studies to have a good impact on the patient profile, by inhibiting tnf- α, by improving mitochondrial function, and good antioxidant action thereby protecting against free radical stress. Its minimal adverse effect profile aside, it also aided in a noticeable improvement in outcome of patients and a reduced need for hepatic replacement therapy. But one Cochrane review ascertained that there was no benefit in using this drug in alcoholic hepatitis. Further studies may be needed before the verdict can be decided either way.

LIVER TRANSPLANTATION:

Finally, liver transplantation can be used as a last resort in those with severe decompensation. The outcomes for transplantation in the setting of alcoholic liver disease are the same if not better than when done for other indications. Thus, although a preliminary half year abstinence is required for any patient to undergo a liver transplant, arguments have been made for transplantation to be a salvage therapy for those, who, otherwise fulfill criteria for transplantation as well as a thorough psychiatric exam.

OTHER THERAPIES:

Attempts have been made to use anabolic androgenic steroids to increase muscle bulk and improve nutritional status. Meta analysis of these trials showed no improved benefit and are thus not recommended. The antithyroid medication Propylthiouracil has been suggested and tried out in patients with alcoholic hepatitis. But no benefit was found. The use of antioxidants, counter intuitively, has not shown any significant benefit. The drugs included in the trial were Vitamin E and Silymarin. But there is hope in the horizon as N-acetylcysteine may be of some benefit in conjunction with corticosteroids. Further studies are needed to establish this idea.

NEWER POTENTIAL TARGETS:

Considering the fact the animals seem to be far more resilient to the development of alcoholic cellular injury and portal hypertension, it has been difficult transposing findings in animal models to clinical practice. Those animal models that have significantly similar injury patterns to live patients with alcoholic liver injury need to be studied deeply. Another factor that needs to be taken into account is the fact that some molecules and cytokines may simply be elevated due to concomitant infection which almost always accompanies alcoholic liver disease and its decompensations. Thus a retrospective approach of finding molecules that are significantly in clinical patients and then using that knowledge to test out the validity of the molecule in a laboratory setting would be prudent. Some stand out instances of such approaches under trial would be:

CXC CHEMOKINES:

Neutrophilic attack is one of the cardinal features of alcoholic liver disease and alcoholic hepatitis. This family of cytokines especially IL-8 and gro- α . Some studies have shown that, not only are these increased in patients with alcoholic hepatitis, but their levels also correlate well with disease activity time and are inversely proportional to survival duration. This is a potential target that must be looked into.

INTERLEUKIN-22:

It belongs to the IL-10 family of cytokines. It is primarily involved with microbe related immunity, a steady state functioning and regeneration. It protects cells from inflammation, oxidative cell stress, and prevents indirect alcohol medicated apoptosis. Considering the target receptors for this molecule are only present on certain cells, its use might be pretty safe. It could also work synergistically with steroids as the increased risk of infection could be counterbalanced by the antimicrobial activity of IL-22.

COMPLEMENT CASCADE INHIBITION:

The activation of complement and complement mediated cell lysis is an important facet of alcohol mediated liver injury. Developing therapies to inhibit the complement cascade may be of help.

MICROFLORA OF THE ALIMENTARY TRACT:

The role of rifaximin in liver disease is now well established. Patients who have a microbial overgrowth consistently have a worse outcome. Similarly the role of LPS in the pathogenesis of alcoholic liver disease is highly suggestive. Thus agents that can antagonize its action at a pre-receptor or a receptor level may well be of benefit.

INHIBITION OF APOPTOSIS:

Multiple trials have suggested the use of caspase inhibitors as a future therapeutic tool in liver diseases where apoptosis of hepatocytes is present extensively, like hepatitis C and steatohepatitis. Similarly it can also be tried in alcoholic liver disease along with IL-22.

BONE MATRIX PROTEINS:

Peculiarly, osteopontin has been found to be expressed in significantly elevated amounts in patients with alcoholic liver disease. Once it can be established that it is a molecule involved in the pathogenesis, it can be a potential target.

ENDOGENOUSLY SYNTHESIZED CANNABINOIDS:

There have been instances where endocannabinoids and their receptors have been upregulated in the setting of Alcoholic liver disease.

NOSTRIN:

Increased nostrin expression may lead to decreased levels of eNOS which leads to the decreased nitric oxide found in alcoholic liver disease patients.

BETAINE

Betaine or trimethyglycine is a substance found in a variety of plant products in nature, originally first isolated from the juice extracted from sugar beet. It may be produced as an intermediary metabolite in the body as well, by the oxidation of choline. It possesses several properties that have made it useful in the treatment of non alcoholicsteatohepatitis across the years, in the form of betaine hydrochloride, which being a substance found across different natural food products, its side effect profile is expected to be extremely favourable. For those reasons, it would appear to be an interesting candidate to be tried for steatosis and liver dysfunction due to other causes as well.

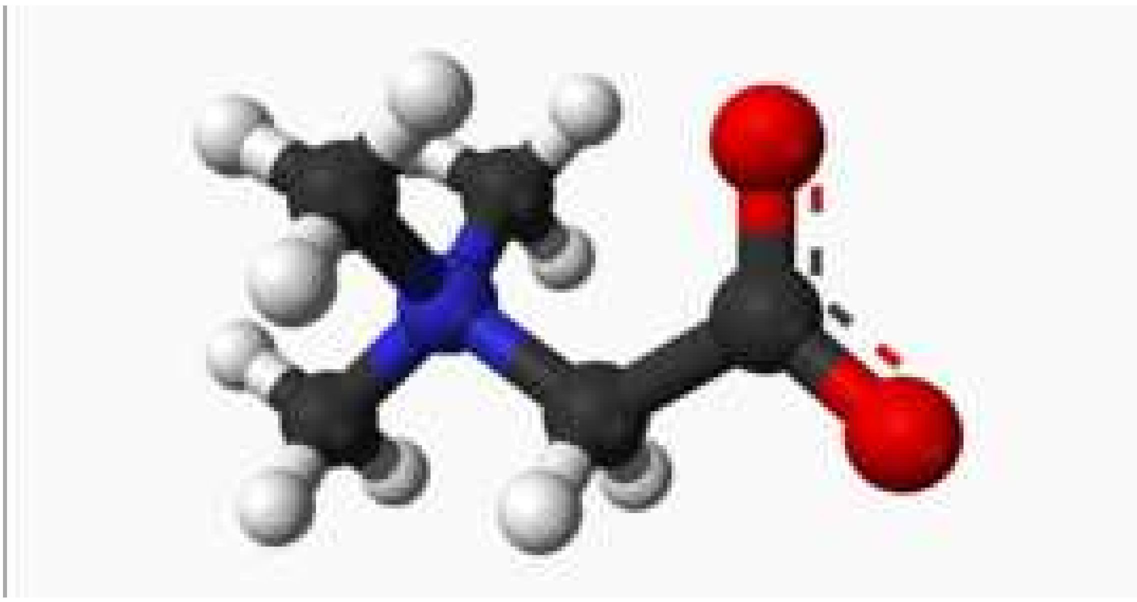


FIGURE - 7

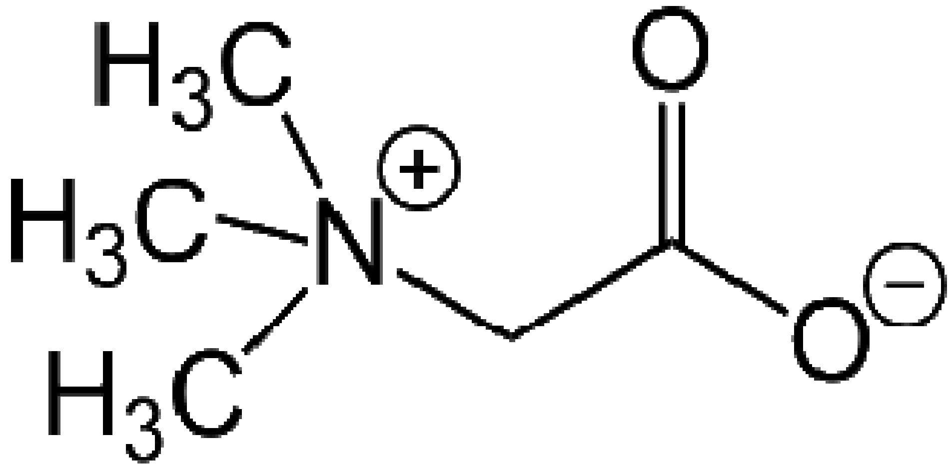


FIGURE - 8

Chemical properties of the drug: Trimethylglycine exists in the form of an N-trimethylated amino acid which makes it a quaternary ammonium compound. It forms a zwitterion at pH 7 - the neutral pH. On reaction with mineral acids, it is converted to TMG of various salts, for example yielding betaine hydrochloride:

$$\mathbf{(CH3)3N^{+}CH2CO2^{-} + HCl \rightarrow [(CH3)3N^{+}CH2CO2H]Cl^{-}}$$

Properties	
Chemical formula	$C_5H_{11}NO_2$
Molar mass	117.146
Appearance	White solid
Melting point	180 °C (356 °F; 453 K) (decomposes)
Solubility in water	Soluble
Solubility	Methanol
Acidity (p*K*a)	1.84

Pharmacological actions of betaine in lower organisms: TMG is an amino acid derived osmolyte that is present in high concentrations (centiosmolars) in many oceanic invertebrates, like crustaceans and molluscs. It serves as a crucial appetitive attractant to generalist non-herbivores such as the carnivorous sea-slug *Pleurobranchaea californica.* TMG is a critical cofactor in methylation, the biochemical reaction that happens in each cell of mammals to create and share methyl groups (CH3) for similar reactions in the cell. These reactions include the production of versatile hormones and autocoids such as dopamine and 5-hydroxytryptamine. Methylation reaction is also used for the synthesis of melatonin and the mitochondrial electron transport chain intermediate coenzyme Q10.The crucial step in the methylation cycle is the remethylation of homocysteine, which will occur through either of two processes. The major cascade is through the enzyme methionine synthase, which requires Cobalamin as a cofactor, and thus also depends indirectly on folic acid and a multitude of other B vitamins. The minor process involves betaine-homocysteinemethyltransferase and uses TMG as a cofactor. Betaine is thus involved in the production of many biologically essential moieties, and may be even more critical in conditions where the major process for the repleting of methionine from homocysteine has been obstructed. Salmon farms use betaine to reduce the osmotic pressure on the fish's cells when they are transferred from freshwater to saltwater.

TMG administration reduces the amount of adipose tissue in pigs

DIETARY SOURCES OF BETAINE:

Betaine is found in a wide variety of naturally occurring foodstuffs like vegetables- beetroot, spinaches and also in various cereals like quinoa and wheat bran.

Food	Betaine (mg/100 g)	Food	Betaine (mg/100 g)
Wheat Bran	1507	Chicken	9
Wheat Germ	1396	Hamburger	9
Spinach	675	Bacon	1
Whole Wheat Bread	180	Brown Rice	0.5
Beets	129	Carrots	0.4
White Bread	102	White Rice	0.3
Sweet Potato	35	Kale	0.3
Cod	10	White Potato	0.2

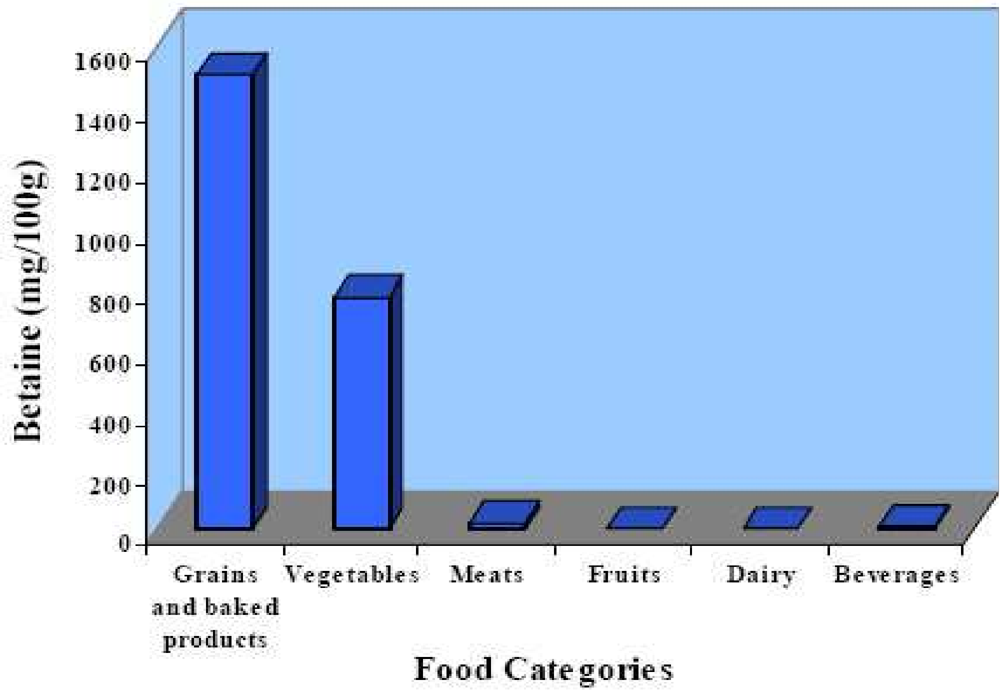


FIGURE - 9

TMG in foods

Food	TMG per 100g
Quinoa	630 mg
Spinach	577 mg
Wheat bran	360 mg
Lamb's quarters	332 mg
Beet	256 mg

CHEMICAL PRODUCTION OF BETAINE:

Producing table sugar from sugar beets offers betaine as byproduct. The amount of betaine contained in sugar beets rivals its sugar content.

Betaine production requires, also, separation by chromatography.

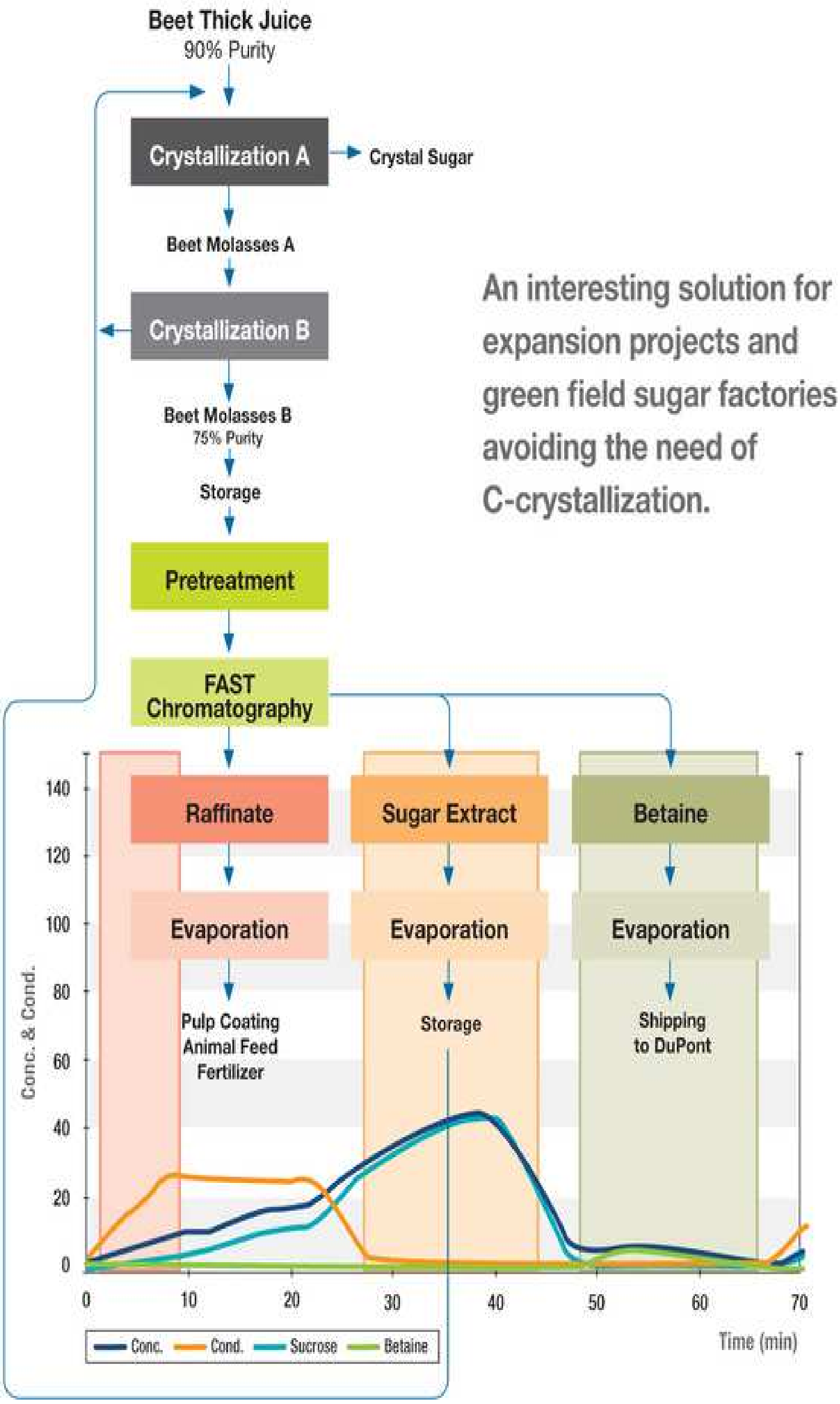


FIGURE - 10

Biosynthesis of betaine in lower organisms: In many organisms, betaine is biosynthesized by oxidation of choline as a two step process. The intermediate, betaine aldehyde, is synthesised by the activity of the mitochondrial enzyme choline oxidase (A.K.A.choline dehydrogenase, EC 1.1.99.1). Betaine aldehyde is subsequently oxidised in the mitochondria of mice to betaine by the enzyme betaine aldehyde dehydrogenase (EC 1.2.1.8). In humans, though, betaine aldehyde activity is undertaken by a nonspecific cytoplasmic aldehyde dehydrogenase (EC 1.2.1.3)

Synthesis of betaine in plants

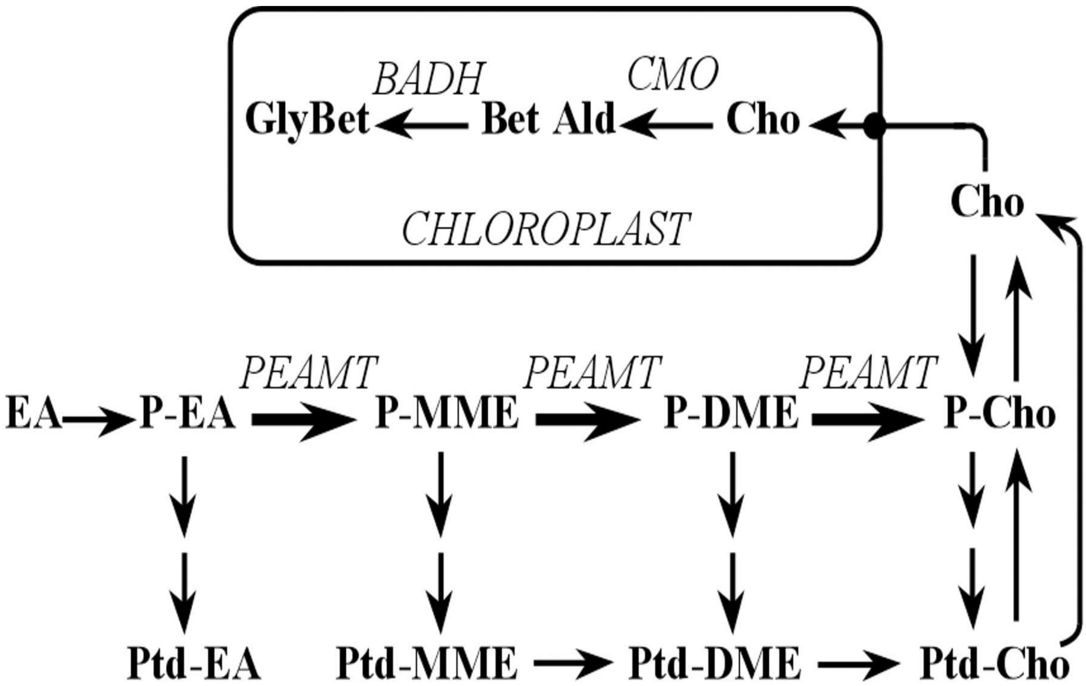


FIGURE - 11

Reactions of betaine in Bacillus subtilis

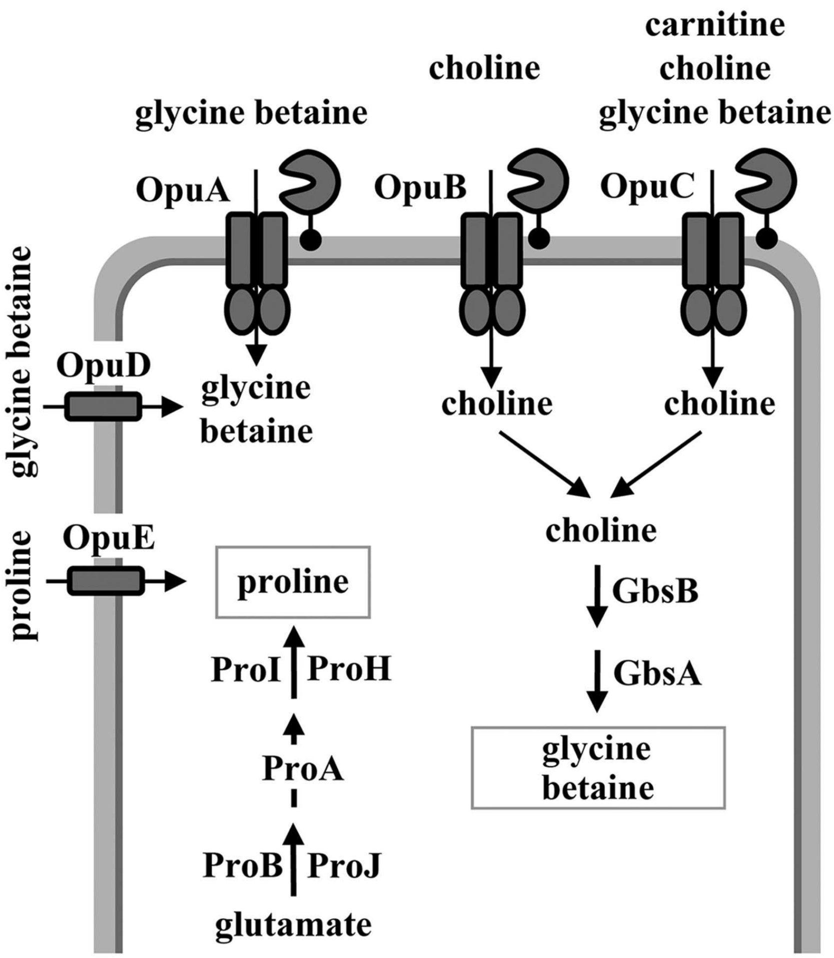


FIGURE - 12

BIOCHEMICAL REACTIONS OF BETAINE:

The reactions driven by betaine leading to its use are: (1) Its ability to act as a methyl group donor in the biochemical reaction for the conversion of to methionine of homocysteine by a methyl transferase; (2) ability to act as a proxy for the ubiquitous methyl group donor for S-adenosylmethionine (SAM) in a reaction where phosphatidylethanolamine in directly methylated to phosphatidyl choline; (3) its protective actions against free radical stress;

(4) it's vital facilitatory role in transsulfuration reactions; (5) metabolic stimulatory effect of adenosine monophosphate -activated protein kinase; and (6) its strong lipotropic property; (7) its ability to be osmotically active.

METHYLATION REACTION AND BETAINE:

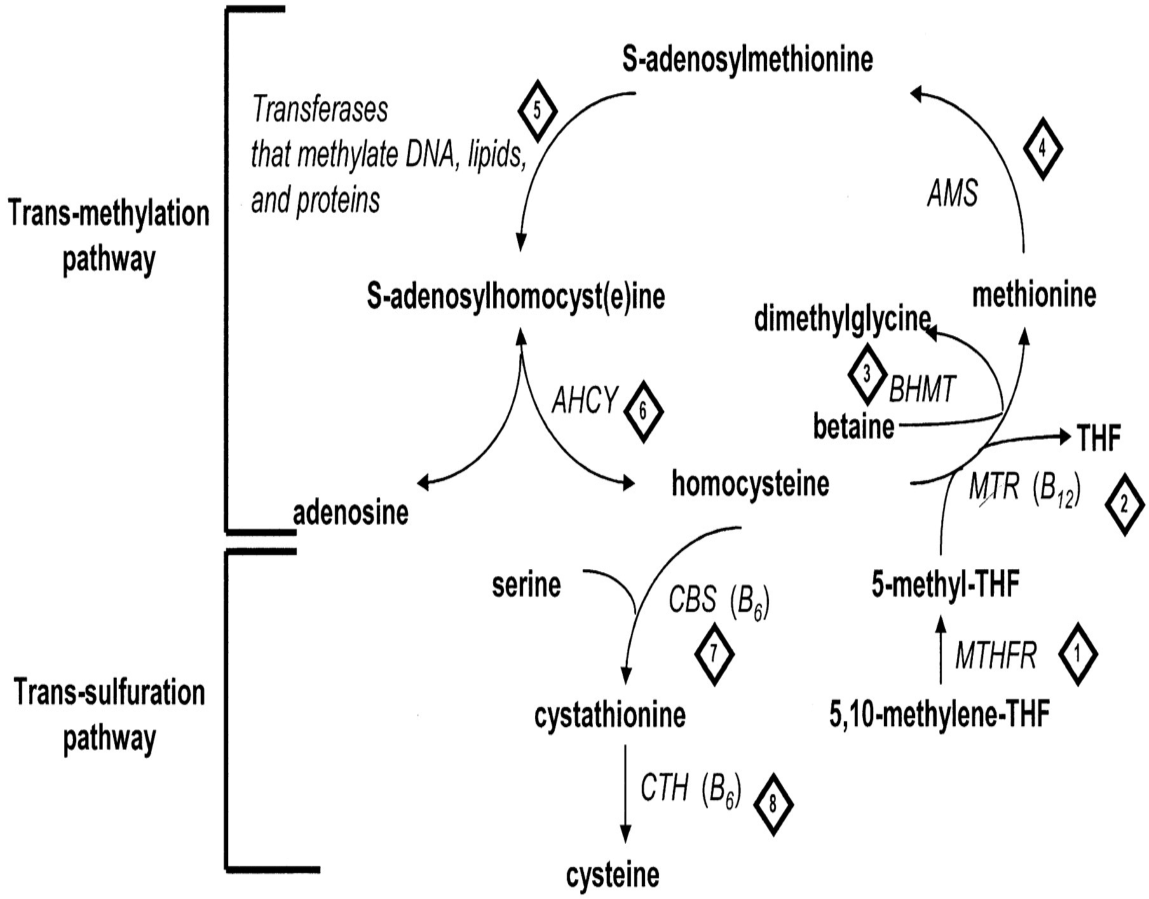


FIGURE - 13

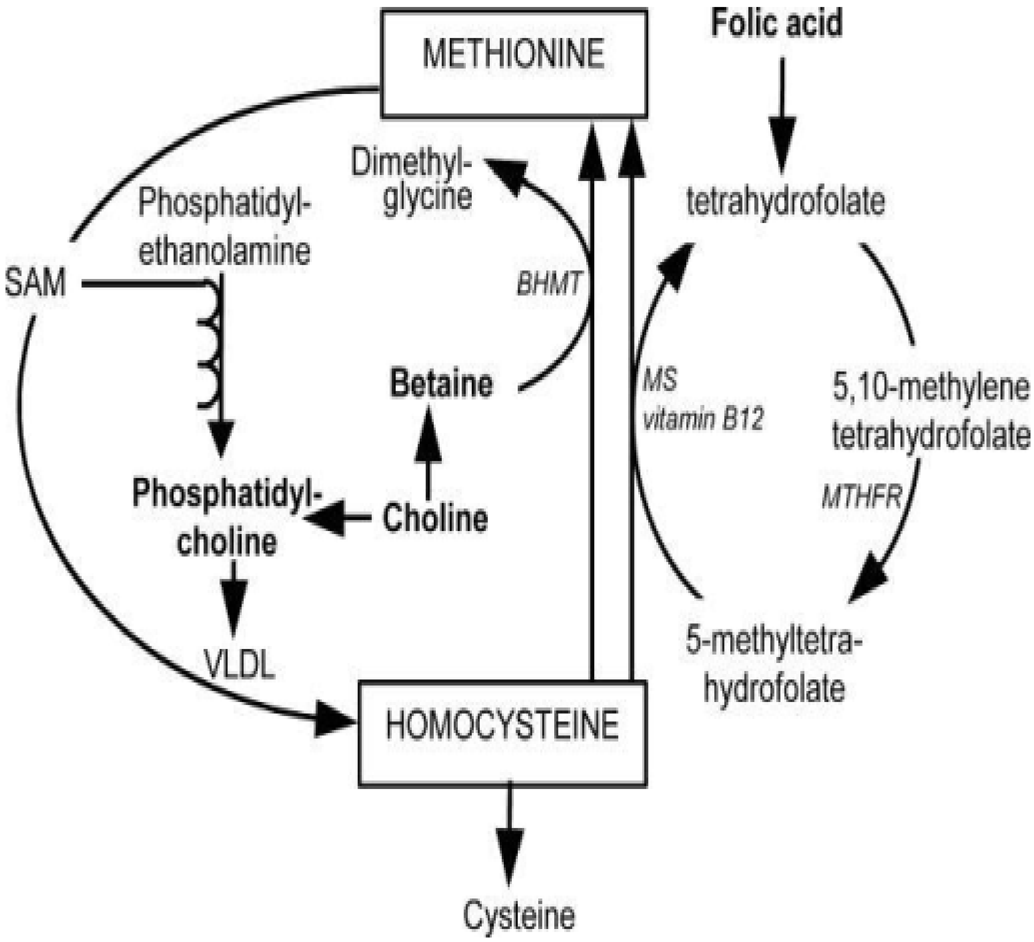


FIGURE - 14

Betaine and its interactions with SAM

Degradation of betaine: TMG upon demethylation reaction would yield dimethylglycine. Complete chemical lysis of TMG yields trimethylamine, the compound giving off the odour of rotten fish.

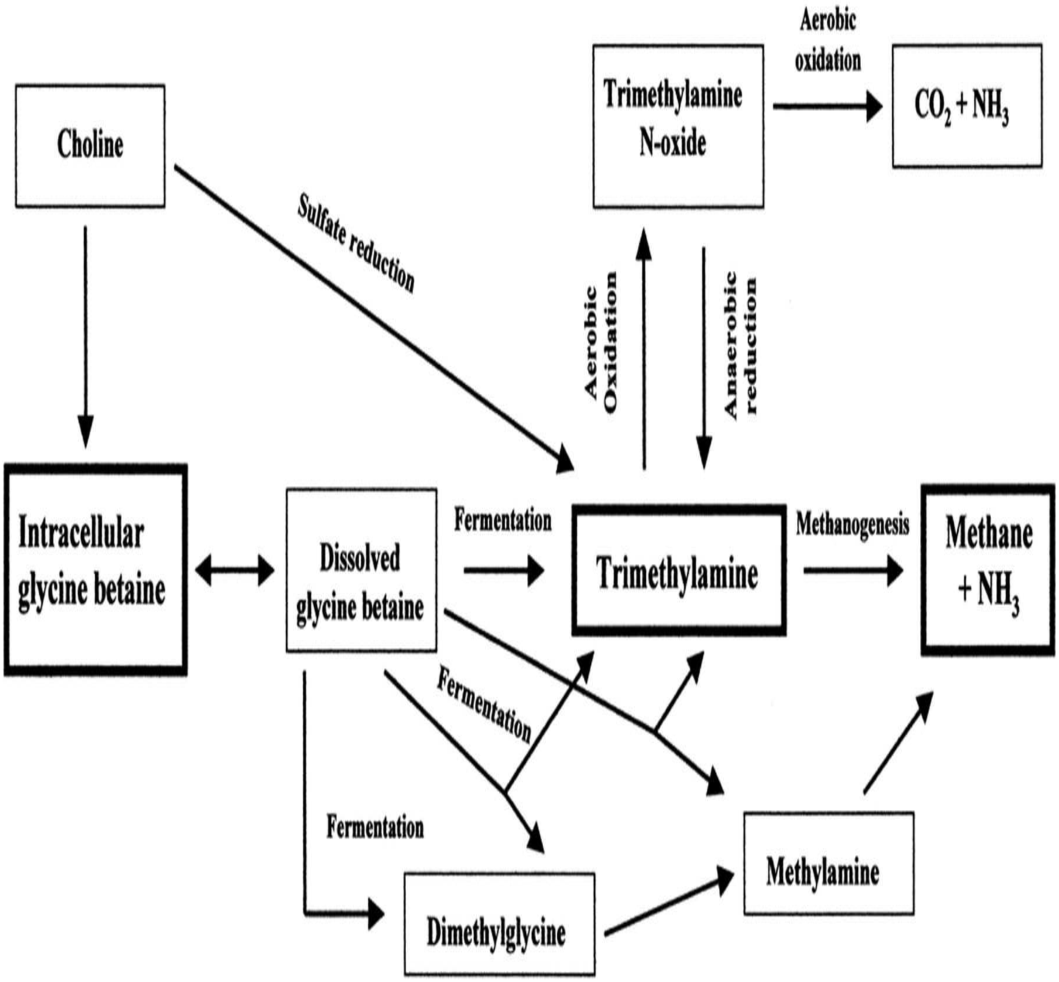


FIGURE - 15

SIDE EFFECT PROFILE:

Patients in previous studies had complained of nausea, bloating, heartburn, constipation, diarrhoea, body odour and itching. Rarely patients also complained of worsening of their peptic ulcer pain. The exact incidences or each adverse reaction was not quantified. No trial reported any serious or fatal adverse reaction to the drug. The drug is currently pregnancy category C and it is not yet known whether the drug is excreted in breast milk. So currently it is not recommended in pregnancy and lactation.

DOSING :

A wide range of doses have been given in previously published studies. The overall dosing range is 6-20g/day in two divided doses. The drug is usually given as a suspension in a solution or water, or as capsules.

Utility in NAFLD:

Betaine, has yet to be tried in the setting of alcoholic liver disease. Upto 2011, four studies of betaine for the treatment of Non alcoholic fatty liver disease had been reported. The first one, a study by Miglio et al could not be considered too seriously as liver biopsy and microscopy was not used to qualify the patients as having non alcoholic fatty liver disease or not, which would put the patient sample set and overall statistical relevance into serious question. The study was published in 2000, being a randomised control trial with double blinding where the patients were given twice a day for eight weeks, oral betaine glucuronate along with diethanolamine glucuronate and nicotinamide ascorbate or placebo. The next trial was by Abdelmalek et al who, in 2001, attempted to use anhydrous betaine on ten patients. The study design was a prospective trial but without a control arm, and so, no blinding the duration of study was for one year out of which three patients did not even finish the trial, although they did experience an improvement of their liver transaminases in their truncated stay in the trial. Three patients would go on to enjoy complete normalization of their transaminases. Three other patients would go on to have improvements while one patient did not respond. Along with the biochemical parameters mentioned above, patients were also subjected to liver biopsies for the purpose of diagnosis and for the purpose of follow up and up- and down-staging of the illness which included degree of steatosis, necroinflammatory grade, and stage of fibrosis after completion of one year of betaine therapy. The size and the lack of placebo, randomization or blinding castes serious aspersions about the validity of the study. Credit, though, still does need to be given as it was a pilot study that paved the way for the rest of us to probe further into this drug. Mukherjee et al between 2003 and 2006 performed a study on thirty five NASH patients who were chosen based on elevated transaminases and liver biopsies being performed within one year of entry into the study and having satisfied the brunt criteria for non alcoholic fatty liver disease, who were then treated with anhydrous betaine ten grams twice a day, for one year. They reported an improvement in transaminases and stage of illness, and a overall better outcome with respect to hepatic fibrosis. Although the study appeared promising, it was a cohort study and there was no control arm or blinding. Abdelmalek et al subsequently reported the results of their second study in may 2010 . The primary aim of this study was to administer ten grams of betaine twice a day and to assess transaminase response. The secondary aim would go on to be determined by histopathology. The total size of this study would include thirty five patients who would complete the trial (seventeen betaine *vs* eighteen placebo), which apart from liver function tests and liver biopsies, also included adipokines levels, basic cytokines, serum homocysteine levels, S-adenosylhomocysteine (SAH), and the liver biopsies were scored according to the Brunt criteria. The study went on to declare that they found no statistically significant improvement in betaine group with respect to the transaminases or the liver biopsies and the improvements seen in either arms of the study were spontaneous and similar. Betaine had shown no appreciable impact on adiponectin, cytokine, and SAH levels. The effect of betaine on the biopsies and histopathology were also placid, with no impact on fibrosis during the study as was claimed by earlier studies. Of even more significance, more patients in the placebo arm had a significant improvement in the steatosis grade (twenty nine percent *vs* sixty one percent, with a ‘p’ value less than 0.01) by more than one point, than the test arm. Slightly confounding though, was the fact that more test patients compared to placebo (seventy one percent *vs* twenty two percent, with a ‘p’ value of less than 0.005) had a stabilized steatosis grade during the study.

The above trial would have categorically meant that betaine should have fallen completely out of favour with researchers in the field of non alcoholic liver disease. But the above studies quoted, with the last one included, do have some expressedly bewildering and sometimes, basic limitations which will, even on retrospect, always lead us to question the underlying validity of each study and its respective outcomes and inferences. It is deeply distressing and confounding to find studies where there is a massive attrition load, especially of more than ten percent. How it would be possible to fill such huge gaps or extrapolate observational results in the setting of such large patient losses was an important question to come up with answers for and rectify. Some observers had commented that since the nature of non alcoholic fatty liver disease was so chronic that perhaps, even improvements would take that long. To add to worries, there exists no clear dosing regimen or efficacy study to ascertain what could be the possible dose required to even attempt using betaine in this setting. The dosages administered in the above trials were simply modified and shoe-horned versions of what the dosing is for homocystinuria for which anhydrous betaine in approved. Inadequacy in knowledge of the drug dosing can mean the drug could've been just right, too little or even too much. For example, study patients in the last mentioned trial had a significantly higher incidence of abdominal related adverse drug reaction (thirty three percent *vs* nine percent, 'p' value less than 0.05), and this only adds to our attrition headache. Perhaps administering protected forms of the drug in the form of capsules or coated preparations, or alternate suspensions of the drug or even development of a newer salt of the same parent drug may be of help. Even more fundamental questions need to be asked about the lack of objective proof of adequate patient compliance during the study.

But for every study that throws up problems and loopholes, there have always been ones where one's curiosity has been kindled. A study on mice by Kathirvel et al showed that betaine reduced fatty change and also helped improve insulin resistance and overall showing some sort of protective benefit in animal models shows why persisting with betaine still makes ample sense. It is obvious that subsequent studies will have to have tighter protocols and more care to prevent confounding factors from obscuring statistical data and assessment of outcomes. These mechanisms of alcoholic liver disease and non alcoholic liver disease do exhibit some common traits in terms of cellular pathogenesis. There has been no idea hitherto, to ascertain the benefits of this drug in the setting of alcohol related disease. Betaine, having demonstrated such a wide variety of biochemical actions in the liver cell, must be experimented with due to its considerably harmless side effect profile and its low cost. The fact that it does not seem to be a hit or miss drug or a drug with a lethal safety margin or any of that sort means that, at the very least, betaine in the future can be used as a great adjunct to a later discovery, if at all it does prove to be unsatisfactory as a mainstay. Other diseases where there has been use of betaine: Homocystinuria, Achlorhydria, Aminoaciduria, poliomyelitis.

MATERIALS

AND

METHODS

MATERIALS AND METHODS

Duration of the Study :

6 months

Study Design:

Randomized Control Study with Single Blinding and Comparison with Placebo

Sample Size:

40 patients

Inclusion Criteria:

1. Patients age >35 with acute on chronic liver disease secondary to alcohol consumption as evidenced by at least one new onset appearance of :-
    a. jaundice, upper GI bleed, hepatic encephalopathy, coagulopathy, spontaneous bacterial peritonitis; in the presence of increased liver enzymes
2. Maddrey score >3
3. Past or present alcohol dependence or withdrawal; or consumption of alcohol > 10 drinks/ week for more than five years at any point in time; or patients with any one criteria positive in the CAGE

questionnaire at any point in time in the present or past.

4. The acute decompensation may be of any cause.

Exclusion Criteria:

1. Age 35 or less
2. Patients in whom the liver disease may be ascribed to any other cause eg. Viral hepatitis, intermediary metabolism defects
3. Patients in whom the maddrey score is below 32
4. Diabetic patients
5. Patients with alcoholic hepatitis without evidence of underlying chronic liver disease

Data Collection and Methods:

Patients selected for clinical study as per inclusion / exclusion criteria are subjected to routine blood investigations like complete hemogram, renal function tests, serum electrolytes, liver function tests, coagulogram, ultrasound of the abdomen, glycemic profile, screening for hepatitis viruses A to E. Detailed clinical examination will be done. Both groups of patients will be subjected to an upper GI endoscopy at the time of inclusion. The cause of that episode of decompensation shall be investigated and duly noted to better subcategorize the response patterns. Test Patients will be given oral therapy with betaine 3g b.d. for 3 months with other standard prescribed drugs while control patients will be given a placebo. Patients will be followed up weekly during the study period where they will be subjected to a full physical exam and routine blood tests in the form of complete blood count, renal and liver function tests, and coagulogram. They will be subjected to a repeat upper GI endoscopy at the end of the study period.

Product / Procedure / Investigation Details:

1. Complete blood count
2. Renal function test
3. Liver function test
4. Coagulogram
5. Detection of markers for viral hepatitis
6. Glycemic Profile
7. Upper GI Endosopy
8. Ultrasonogram of the abdomen

Ethical Committee approval : Obtained

OBSERVATION

AND

RESULTS

OBSERVATION AND RESULTS

This study was done to determine the effects on betaine therapy on acute on chronic alcoholic liver disease. 40 patients were chosen under the inclusion criteria and 20 of them were subjected to the drug while 20 were subjected to placebo therapy. The following data was obtained based on the data collected as part of the study:

TABLE 1 : AGE OF SUBJECTS

		AGE					
		Mean	**Median**	**Minimum**	**Maximum**	**Standard Deviation**	**Standard Error of Mean**
group	Experiment	54.45	56.00	41.00	63.00	7.06	1.58
	Control	54.30	54.00	39.00	68.00	7.55	1.69

$p>0.05$, No difference between two groups among 2 groups

TOTAL BILIRUBIN:

TABLE 2 : MEASURE: T_BILIRUBIN

group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	13.51	.485	12.529	14.491
	2	12.46	.194	12.071	12.856
	3	12.55	.216	12.113	12.988
	4	12.59	.303	11.985	13.211
	5	12.25	.269	11.713	12.802
	6	9.11	.787	7.517	10.705
	7	7.39	.513	6.353	8.428
	8	6.92	.247	6.429	7.428
	9	6.60	.198	6.204	7.005
	10	3.15	.214	2.725	3.591
	11	3.08	.201	2.678	3.494
	12	3.42	.367	2.681	4.168
	13	3.39	.346	2.689	4.090
Control	1	12.38	.485	11.399	13.361
	2	12.46	.194	12.075	12.861
	3	13.05	.216	12.612	13.487
	4	13.11	.303	12.503	13.728
	5	13.18	.269	12.643	13.732
	6	11.28	.787	9.687	12.875
	7	7.43	.513	6.399	8.474
	8	6.87	.247	6.377	7.377
	9	6.66	.198	6.263	7.064
	10	3.78	.214	3.354	4.220
	11	3.13	.201	2.724	3.540
	12	3.52	.367	2.785	4.272
	13	3.52	.346	2.823	4.225

PAIRWISE COMPARISONS

TABLE 3 : MEASURE: T_BILIRUBIN

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	-.304	.175	.091	-.659	.051
Control	Experiment	.304	.175	.091	-.051	.659

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE 4 : DIRECT BILIRUBIN

4. group * WEEKS					
Measure: d_billrubin					
group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	13.51	.485	12.529	14.491
	2	12.46	.194	12.071	12.856
	3	12.55	.216	12.113	12.988
	4	12.59	.303	11.985	13.211
	5	12.25	.269	11.713	12.802
	6	9.11	.787	7.517	10.705
	7	7.39	.513	6.353	8.428
	8	6.92	.247	6.429	7.428
	9	6.60	.198	6.204	7.005
	10	3.15	.214	2.725	3.591
	11	3.08	.201	2.678	3.494
	12	3.42	.367	2.681	4.168
	13	3.39	.346	2.689	4.090
Control	1	12.38	.485	11.399	13.361
	2	12.46	.194	12.075	12.861
	3	13.05	.216	12.612	13.487
	4	13.11	.303	12.503	13.728
	5	13.18	.269	12.643	13.732
	6	11.28	.787	9.687	12.875
	7	7.43	.513	6.399	8.474
	8	6.87	.247	6.377	7.377
	9	6.66	.198	6.263	7.064
	10	3.78	.214	3.354	4.220
	11	3.13	.201	2.724	3.540
	12	3.52	.367	2.785	4.272
	13	3.52	.346	2.823	4.225

PAIRWISE COMPARISONS

TABLE 5 : MEASURE: D_BILIRUBIN

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference	
					Lower Bound	Upper Bound
Experiment	Control	-.304	.175	.091	-.659	.051
Control	Experiment	.304	.175	.091	-.051	.659

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

LIVER ENZYME- SGPT

TABLE 6 : SGPT

Measure: pt_					
group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	170.850	9.143	152.340	189.360
	2	176.600	3.165	170.192	183.008
	3	172.300	3.404	165.409	179.191
	4	170.650	3.292	163.986	177.314
	5	175.900	4.367	167.059	184.741
	6	131.100	5.632	119.698	142.502
	7	126.700	3.349	119.920	133.480
	8	120.350	3.302	113.665	127.035
	9	124.500	3.357	117.704	131.296
	10	70.400	3.286	63.748	77.052
	11	83.100	6.289	70.369	95.831
	12	78.700	4.812	68.959	88.441
	13	74.050	3.594	66.774	81.326
Control	1	156.950	9.143	138.440	175.460
	2	174.850	3.165	168.442	181.258
	3	176.450	3.404	169.559	183.341
	4	177.600	3.292	170.936	184.264
	5	177.500	4.367	168.659	186.341
	6	123.250	5.632	111.848	134.652
	7	122.500	3.349	115.720	129.280
	8	124.300	3.302	117.615	130.985
	9	130.500	3.357	123.704	137.296
	10	70.100	3.286	63.448	76.752
	11	66.250	6.289	53.519	78.981
	12	67.600	4.812	57.859	77.341
	13	67.250	3.594	59.974	74.526

PAIRWISE COMPARISONS

TABLE 7 : PAIRWISE COMPARISONS

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	3.085	2.143	.158	-1.254	7.423
Control	Experiment	-3.085	2.143	.158	-7.423	1.254

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE 8 Liver Enzyme- SGOT

4. group * WEEKS					
Measure: ot_					
group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	394.500	12.410	369.377	419.623
	2	333.650	20.168	292.822	374.478
	3	318.600	17.889	282.385	354.815
	4	376.500	18.115	339.829	413.171
	5	325.500	19.006	287.024	363.976
	6	199.550	9.838	179.633	219.467

	7	187.150	1.767	183.573	190.727
	8	187.650	1.770	184.066	191.234
	9	187.950	1.639	184.633	191.267
	10	63.350	3.704	55.852	70.848
	11	77.250	12.814	51.309	103.191
	12	71.900	10.878	49.879	93.921
	13	72.200	6.645	58.748	85.652
Control	1	386.350	12.410	361.227	411.473
	2	347.700	20.168	306.872	388.528
	3	322.950	17.889	286.735	359.165
	4	348.150	18.115	311.479	384.821
	5	353.700	19.006	315.224	392.176
	6	195.450	9.838	175.533	215.367
	7	186.700	1.767	183.123	190.277
	8	187.250	1.770	183.666	190.834
	9	189.000	1.639	185.683	192.317
	10	69.850	3.704	62.352	77.348
	11	77.750	12.814	51.809	103.691
	12	87.400	10.878	65.379	109.421
	13	72.300	6.645	58.848	85.752

Pairwise Comparisons

TABLE NO . 9 Measure: ot_

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	-2.215	4.993	.660	-12.323	7.892
Control	Experiment	2.215	4.993	.660	-7.892	12.323

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO. 10 Liver Enzyme Alkaline Phosphatase

4. group * WEEKS					
Measure: alp_					
group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	62.700	2.553	57.531	67.869
	2	59.500	1.585	56.291	62.709
	3	61.500	1.433	58.598	64.402
	4	59.350	1.354	56.610	62.090
	5	60.500	1.379	57.707	63.293
	6	60.650	1.132	58.359	62.941
	7	60.850	1.241	58.337	63.363
	8	60.850	1.354	58.109	63.591
	9	59.850	1.602	56.608	63.092
	10	60.350	1.371	57.574	63.126
	11	62.050	1.321	59.377	64.723
	12	57.950	1.601	54.708	61.192
	13	59.600	1.325	56.918	62.282
Control	1	61.400	2.553	56.231	66.569
	2	61.400	1.585	58.191	64.609
	3	58.350	1.433	55.448	61.252
	4	62.000	1.354	59.260	64.740
	5	60.800	1.379	58.007	63.593
	6	59.150	1.132	56.859	61.441
	7	59.700	1.241	57.187	62.213
	8	60.550	1.354	57.809	63.291
	9	56.450	1.602	53.208	59.692
	10	62.400	1.371	59.624	65.176
	11	59.650	1.321	56.977	62.323
	12	61.300	1.601	58.058	64.542
	13	59.500	1.325	56.818	62.182

Pairwise Comparisons

TABLE NO.11 Measure: alp_

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	.235	.728	.749	-1.240	1.709
Control	Experiment	-.235	.728	.749	-1.709	1.240

Based on estimated marginal means
a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO.12 Total Protein:

4. group * WEEKS					
Measure: t_protein					
group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	5.51	.052	5.405	5.615
	2	5.21	.051	5.112	5.319
	3	5.18	.045	5.090	5.271
	4	5.26	.048	5.165	5.358
	5	5.27	.058	5.159	5.395
	6	5.23	.049	5.136	5.336
	7	5.20	.054	5.099	5.316
	8	5.25	.050	5.149	5.353
	9	5.16	.055	5.050	5.272
	10	5.20	.044	5.117	5.294
	11	5.20	.054	5.097	5.315
	12	5.20	.043	5.115	5.290
	13	5.26	.046	5.171	5.359

Control	1	5.52	.052	5.415	5.625
	2	5.13	.051	5.033	5.240
	3	5.09	.045	5.006	5.187
	4	5.20	.048	5.107	5.300
	5	5.17	.058	5.059	5.296
	6	5.19	.049	5.090	5.290
	7	5.13	.054	5.025	5.242
	8	5.25	.050	5.157	5.361
	9	5.19	.055	5.086	5.309
	10	5.25	.044	5.167	5.344
	11	5.06	.054	4.953	5.171
	12	5.17	.043	5.085	5.260
	13	5.11	.046	5.017	5.205

Pairwise Comparisons

TABLE NO.13 Measure: t_protein

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
Experiment	Control	.051*	.023	.032	.005	.098
Control	Experiment	-.051*	.023	.032	-.098	-.005

Based on estimated marginal means
*. The mean difference is significant at the .05 level.
b. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO.14 Serum Albumin:

4. group * WEEKS					
Measure: s_albumin					
group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	2.61	.034	2.547	2.685
	2	2.58	.026	2.534	2.640
	3	2.58	.025	2.533	2.636
	4	2.57	.027	2.519	2.629
	5	2.60	.029	2.545	2.663
	6	2.60	.027	2.550	2.658
	7	2.60	.023	2.561	2.654
	8	2.62	.026	2.571	2.675
	9	2.55	.027	2.501	2.611
	10	2.60	.030	2.546	2.667
	11	2.54	.024	2.499	2.596
	12	2.60	.022	2.563	2.653
	13	2.58	.028	2.529	2.644
Control	1	2.58	.033	2.518	2.652
	2	2.54	.026	2.497	2.600
	3	2.55	.025	2.503	2.603
	4	2.60	.026	2.553	2.660
	5	2.59	.028	2.536	2.652
	6	2.57	.026	2.525	2.630
	7	2.63	.022	2.592	2.683
	8	2.60	.025	2.550	2.651
	9	2.62	.027	2.569	2.677
	10	2.59	.029	2.538	2.656
	11	2.53	.023	2.487	2.582
	12	2.54	.022	2.500	2.588
	13	2.61	.028	2.557	2.669

TABLE NO.15 Measure: s_albumin

Pairwise Comparisons						
(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	.007	.012	.551	-.017	.031
Control	Experiment	-.007	.012	.551	-.031	.017
Based on estimated marginal means						
a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).						

p>0.05 no difference between group and weeks

INR:

TABLE NO.16 INR

4. group * WEEKS					
Measure: inr_					
group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	4.35	.102	4.143	4.557
	2	3.48	.097	3.288	3.681
	3	3.55	.073	3.403	3.698
	4	1.99	.123	1.747	2.244
	5	2.31	.174	1.966	2.670
	6	2.02	.100	1.821	2.225
	7	1.89	.064	1.769	2.027
	8	2.01	.068	1.874	2.151
	9	2.06	.060	1.939	2.182
	10	1.87	.071	1.729	2.017
	11	2.00	.083	1.836	2.174
	12	1.98	.075	1.831	2.136
	13	2.02	.087	1.844	2.197

Control	1	3.96	.102	3.753	4.167
	2	3.63	.097	3.436	3.830
	3	3.60	.073	3.453	3.748
	4	2.15	.123	1.903	2.400
	5	2.30	.174	1.956	2.660
	6	2.05	.100	1.850	2.253
	7	1.86	.064	1.739	1.997
	8	1.99	.068	1.858	2.135
	9	2.09	.060	1.973	2.216
	10	1.94	.071	1.797	2.085
	11	2.03	.083	1.867	2.205
	12	2.02	.075	1.876	2.182
	13	2.08	.087	1.904	2.257

TABLE NO.17 PAIRWISE COMPARISONS

Pairwise Comparisons						
(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	-.013	.036	.714	-.087	.060
Control	Experiment	.013	.036	.714	-.060	.087
Based on estimated marginal means						
a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).						

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO.18 HAEMOGLOBIN

Haemoglobin:

4. group * WEEKS						
Measure: HB_						
group		WEEKS	Mean	Std. Error	95% Confidence Interval	
					Lower Bound	Upper Bound
Experiment		1	8.31	.306	7.691	8.929
		2	8.42	.327	7.763	9.087
		3	7.92	.322	7.273	8.577
		4	8.14	.320	7.492	8.788
		5	7.96	.319	7.314	8.606
		6	8.48	.322	7.834	9.136
		7	7.77	.319	7.125	8.415
		8	7.67	.319	7.030	8.320
		9	8.16	.320	7.512	8.808
		10	7.41	.320	6.766	8.064
	P<0.05	11	7.50	.320	6.858	8.152
		12	7.14	.325	6.483	7.797
		13	7.33	.323	6.676	7.984
Control		1	7.96	.306	7.346	8.584
		2	7.85	.327	7.193	8.517
		3	7.38	.322	6.728	8.032
		4	7.66	.320	7.017	8.313
		5	7.47	.319	6.829	8.121
		6	7.95	.322	7.299	8.601
		7	7.28	.319	6.640	7.930
		8	7.19	.319	6.545	7.835
		9	7.66	.320	7.017	8.313
		10	6.90	.320	6.256	7.554
	P<0.05	11	7.00	.320	6.353	7.647
		12	6.62	.325	5.963	7.277
		13	6.81	.323	6.156	7.464

TABLE NO.19 HB

Pairwise Comparisons

Measure: HB_

(I) group	(J) group	Mean Difference (I-J)	Std. Error	P VALUE	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	.498	.449	.274	-.411	1.407
Control	Experiment	-.498	.449	.274	-1.407	.411

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO.20 PACKED CELL VOLUME

Packed Cell Volume:

4. group * WEEKS

Measure: PCV_

group		WEEKS	Mean	Std. Error	95% Confidence Interval	
					Lower Bound	Upper Bound
Experiment	P<0.05	1	26.30	.743	24.797	27.803
		2	24.20	.612	22.960	25.440
		3	23.35	.583	22.170	24.530
		4	23.45	.730	21.971	24.929
		5	21.70	.622	20.440	22.960
		6	23.80	.688	22.407	25.193
		7	22.70	.664	21.355	24.045
		8	23.15	.678	21.777	24.523
		9	23.55	.656	22.222	24.878
		10	22.35	.667	20.999	23.701
		11	21.40	.604	20.177	22.623

		12	22.75	.668	21.398	24.102
		13	22.75	.708	21.317	24.183
Control	P<0.05	1	25.45	.743	23.947	26.953
		2	23.10	.612	21.860	24.340
		3	22.10	.583	20.920	23.280
		4	22.35	.730	20.871	23.829
		5	23.70	.622	22.440	24.960
		6	23.85	.688	22.457	25.243
		7	23.20	.664	21.855	24.545
		8	23.20	.678	21.827	24.573
		9	21.70	.656	20.372	23.028
		10	23.00	.667	21.649	24.351
		11	23.35	.604	22.127	24.573
		12	21.80	.668	20.448	23.152
		13	22.55	.708	21.117	23.983

TABLE NO.21 PCV

Pairwise Comparisons

Measure: PCV_

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	.162	.417	.700	-.682	1.005
Control	Experiment	-.162	.417	.700	-1.005	.682

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO.22 WHITE CELL COUNT

Total White Cell Count:

4. group * week						
Measure: TC_						
group		week	Mean	Std. Error	95% Confidence Interval	
					Lower Bound	Upper Bound
Experiment		1	10.510	.805	8.881	12.139
		2	7.352	.442	6.458	8.246
		3	7.545	.454	6.625	8.465
		4	7.394	.393	6.598	8.191
		5	8.393	.441	7.500	9.286
		6	7.713	.429	6.843	8.582
		7	7.499	.426	6.636	8.363
		8	7.258	.451	6.344	8.172
		9	7.750	.491	6.755	8.744
		10	6.827	.492	5.830	7.824
		11	7.816	.416	6.973	8.659
	P<0.05	12	7.915	.499	6.905	8.926
		13	7.892	.452	6.978	8.807
Control		1	12.035	.805	10.406	13.664
		2	7.771	.442	6.878	8.665
		3	7.644	.454	6.724	8.564
		4	7.706	.393	6.910	8.503
		5	7.202	.441	6.310	8.095
		6	7.988	.429	7.118	8.857
		7	7.053	.426	6.190	7.916
		8	7.136	.451	6.222	8.049
		9	7.052	.491	6.058	8.047
		10	7.753	.492	6.756	8.750
		11	7.292	.416	6.450	8.135
	P<0.05	12	7.518	.499	6.508	8.529
		13	7.526	.452	6.611	8.440

TABLE NO.23 TC

Pairwise Comparisons

Measure: TC_

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	.014	.215	.947	-.420	.449
Control	Experiment	-.014	.215	.947	-.449	.420

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANT DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO.24 PLATELET COUNT

Platelet Count:

Measure: PLATELET						
group	WEEKS	Mean	Std. Error	95% Confidence Interval		P VALUE
				Lower Bound	Upper Bound	
	1	54200	4050	46002	62398	
	2	53150	4018	45015	61285	
	3	55150	3980	47092	63208	
	4	60150	4047	51958	68342	
	5	51200	3960	43183	59217	
	6	45150	3882	37291	53009	
	7	46150	3895	38265	54035	
	8	61200	4083	52934	69466	
	9	52150	3901	44252	60048	
	10	47150	3907	39240	55060	
Experiment	11	53150	4002	45048	61252	P<0.05

	12	56150	4540	46959	65341	
	13	59150	4036	50979	67321	
Control	1	70100	4050	61902	78298	P<0.05
	2	67950	4018	59815	76085	
	3	69300	3980	61242	77358	
	4	74400	4047	66208	82592	
	5	65800	3960	57783	73817	
	6	61350	3882	53491	69209	
	7	61600	3895	53715	69485	
	8	73980	4083	65714	82246	
	9	66880	3901	58982	74778	
	10	62330	3907	54420	70240	
	11	66930	4002	58828	75032	
	12	65735	4540	56544	74926	
	13	72000	4036	63829	80171	

TABLE NO.25 PLATELET

Pairwise Comparisons						
Measure: PLATELET						
(I) group	(J) group	Mean Difference (I-J)	Std. Error	P VALUE	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
Experiment	Control	-14173.458*	5571.545	.15	-25452.460	-2894.455
Control	Experiment	14173.458*	5571.545	.15	2894.455	25452.460
Based on estimated marginal means						
*. The mean difference is significant at the .05 level.						
b. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).						

THE ABOVE TABLES DEPICTS THERE IS NO SIGNIFICANT DIFFERENCE BETWEEN WEEKS AND EXPERIMENTAL AND CONTROL GROUP

TABLE NO.26 BLOOD UREA

Blood Urea:

4. group * WEEKS						
Measure: UREA_						
group		WEEKS	Mean	Std. Error	95% Confidence Interval	
					Lower Bound	Upper Bound
Experiment	P< 0.05	1	52.350	2.590	47.107	57.593
		2	43.650	1.947	39.708	47.592
		3	45.200	1.966	41.221	49.179
		4	42.700	1.591	39.480	45.920
		5	38.750	1.546	35.620	41.880
		6	40.550	1.766	36.975	44.125
		7	42.800	1.464	39.837	45.763
		8	44.250	1.815	40.575	47.925
		9	40.700	1.788	37.081	44.319
		10	41.300	1.659	37.942	44.658
		11	41.000	1.707	37.544	44.456
		12	43.300	1.879	39.495	47.105
		13	38.450	1.583	35.246	41.654
Control	P< 0.05	1	48.200	2.590	42.957	53.443
		2	40.400	1.947	36.458	44.342
		3	42.950	1.966	38.971	46.929
		4	41.350	1.591	38.130	44.570
		5	41.850	1.546	38.720	44.980
		6	43.100	1.766	39.525	46.675
		7	40.500	1.464	37.537	43.463
		8	42.150	1.815	38.475	45.825
		9	43.550	1.788	39.931	47.169
		10	43.050	1.659	39.692	46.408
		11	41.050	1.707	37.594	44.506
		12	41.700	1.879	37.895	45.505
		13	40.500	1.583	37.296	43.704

TABLE NO.27 UREA

Pairwise Comparisons

Measure: UREA_

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	.358	.803	.658	-1.267	1.983
Control	Experiment	-.358	.803	.658	-1.983	1.267

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNGICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO.28 SERUM CREATINE

Serum Creatine :

4. group * WEEKS

Measure: creatine_

group		WEEKS	Mean	Std. Error	95% Confidence Interval	
					Lower Bound	Upper Bound
Experiment	p>0.05	1	1.23	.058	1.117	1.353
		2	1.21	.034	1.143	1.278
		3	1.19	.035	1.126	1.269
		4	1.22	.035	1.159	1.300
		5	1.27	.035	1.207	1.350
		6	1.21	.041	1.137	1.302
		7	1.17	.045	1.086	1.270
		8	1.20	.047	1.112	1.301
		9	1.18	.052	1.078	1.289
		10	1.24	.034	1.179	1.315

		11	1.26	.033	1.199	1.332
		12	1.21	.037	1.144	1.292
		13	1.23	.034	1.165	1.301
Control	p> 0.05	1	1.04	.058	.922	1.158
		2	1.21	.034	1.143	1.279
		3	1.27	.035	1.203	1.346
		4	1.19	.035	1.120	1.261
		5	1.26	.035	1.193	1.336
		6	1.25	.041	1.167	1.332
		7	1.24	.045	1.152	1.336
		8	1.24	.047	1.153	1.341
		9	1.22	.052	1.115	1.325
		10	1.22	.034	1.154	1.290
		11	1.19	.033	1.132	1.264
		12	1.25	.037	1.185	1.333
		13	1.22	.034	1.159	1.296

THE ABOVE TABLES DEPICTS THERE IS no SIGNIFICANCE DIFFERENCE BETWEEN WEEKS EXPERIMENTAL AND CONTROL GROUP

TABLE NO.29 SODIUM

4. group * WEEKS					
Measure: sodium_					
group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	133.600	1.464	130.636	136.564
	2	131.900	.683	130.517	133.283
	3	132.400	.710	130.962	133.838
	4	130.700	.780	129.122	132.278
	5	131.750	.732	130.269	133.231
	6	130.450	.748	128.937	131.963
	7	132.400	.678	131.027	133.773
	8	130.350	.729	128.873	131.827
	9	132.700	.746	131.191	134.209
	10	131.700	.657	130.370	133.030
	11	133.000	.604	131.778	134.222
	12	132.750	.804	131.123	134.377

	13	132.600	.763	131.054	134.146
Control	1	135.300	1.464	132.336	138.264
	2	131.600	.683	130.217	132.983
	3	132.150	.710	130.712	133.588
	4	130.750	.780	129.172	132.328
	5	131.500	.732	130.019	132.981
	6	132.250	.748	130.737	133.763
	7	132.850	.678	131.477	134.223
	8	132.750	.729	131.273	134.227
	9	132.300	.746	130.791	133.809
	10	131.900	.657	130.570	133.230
	11	132.050	.604	130.828	133.272
	12	131.950	.804	130.323	133.577
	13	131.700	.763	130.154	133.246

TABLE NO.30 SODIUM

Pairwise Comparisons

Measure: sodium_

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	-.212	.334	.530	-.888	.465
Control	Experiment	.212	.334	.530	-.465	.888

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO.31 POTASSIUM

Potassium:

4. group * WEEKS					
Measure: potassium_					
group	WEEKS	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Experiment	1	5.42	1.333	2.722	8.118
	2	3.43	.165	3.099	3.766
	3	3.43	.159	3.108	3.752
	4	3.45	.152	3.148	3.763
	5	3.56	.145	3.274	3.861
	6	3.55	.158	3.233	3.874
	7	3.43	.136	3.159	3.709
	8	3.58	.159	3.264	3.909
	9	3.65	.136	3.379	3.931
	10	3.57	.152	3.267	3.882
	11	3.52	.135	3.249	3.796
	12	3.50	.213	3.072	3.933
	13	3.33	.144	3.047	3.630
Control	1	3.76	1.333	1.067	6.463
	2	3.65	.165	3.321	3.988
	3	3.62	.159	3.303	3.947
	4	3.92	.152	3.621	4.236
	5	3.58	.145	3.290	3.878
	6	3.59	.158	3.273	3.914
	7	3.71	.136	3.436	3.987
	8	3.56	.159	3.244	3.889
	9	3.41	.136	3.139	3.690
	10	3.35	.152	3.048	3.662
	11	3.48	.135	3.209	3.756
	12	3.58	.213	3.150	4.011
	13	3.40	.144	3.116	3.698

TABLE NO.32 POTASSIUM

Pairwise Comparisons

Measure: potassium_

(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Experiment	Control	.062	.149	.681	-.240	.363
Control	Experiment	-.062	.149	.681	-.363	.240

THE ABOVE TABLES DEPICTS THERE IS SIGNIFICANCE DIFFERENCE BETWEEN WEEKS . BUT NOT IN EXPERIMENTAL AND CONTROL GROUP

TABLE NO.33 ENDOSCOPY

Endoscopic findings: At Admission:

Crosstab

Count

		UPPER_GI_ENDOSCOPY			Total
		GRADE 1 EV	GRADE 2 EV	GRADE 3 EV	
group	Experiment	0	15	5	20
	Control	1	15	4	20
Total		1	30	9	40

After 12 weeks:

TABLE NO.34 ENDOSCOPY

Crosstab

Count

		UGI_ENDOSCOPY_WEEK_12			Total
		GRADE 1 EV	GRADE 2 EV	GRADE 3 EV	
group	Experiment	0	15	5	20
	Control	1	14	5	20
Total		1	29	10	40

TABLE NO.35 CAUSE OF ACUTE EPISODE

Cause of Current Acute episode:

Crosstab

Count

		CAUSE_OF_HEPATITIS		Total
		ALCOHOL INTAKE	Portal Vein Thrombosis	
group	Experiment	18	2	20
	Control	18	2	20
Total		36	4	40

TABLE NO.36 GLYCEMIC PROFILE

Glycemic profile:

Crosstab

Count

		GLYCAEMIC_PROFILE		Total
		ABNORMAL	NORMAL	
group	Experiment	0	20	20
	Control	0	20	20
Total		0	40	40

For this data the following statistical analysis was used

Repeated measures Anova (comparisons of 13 weeks with 2 groups)

Independent t test for compare 2 groups for age (Experiment and Control)

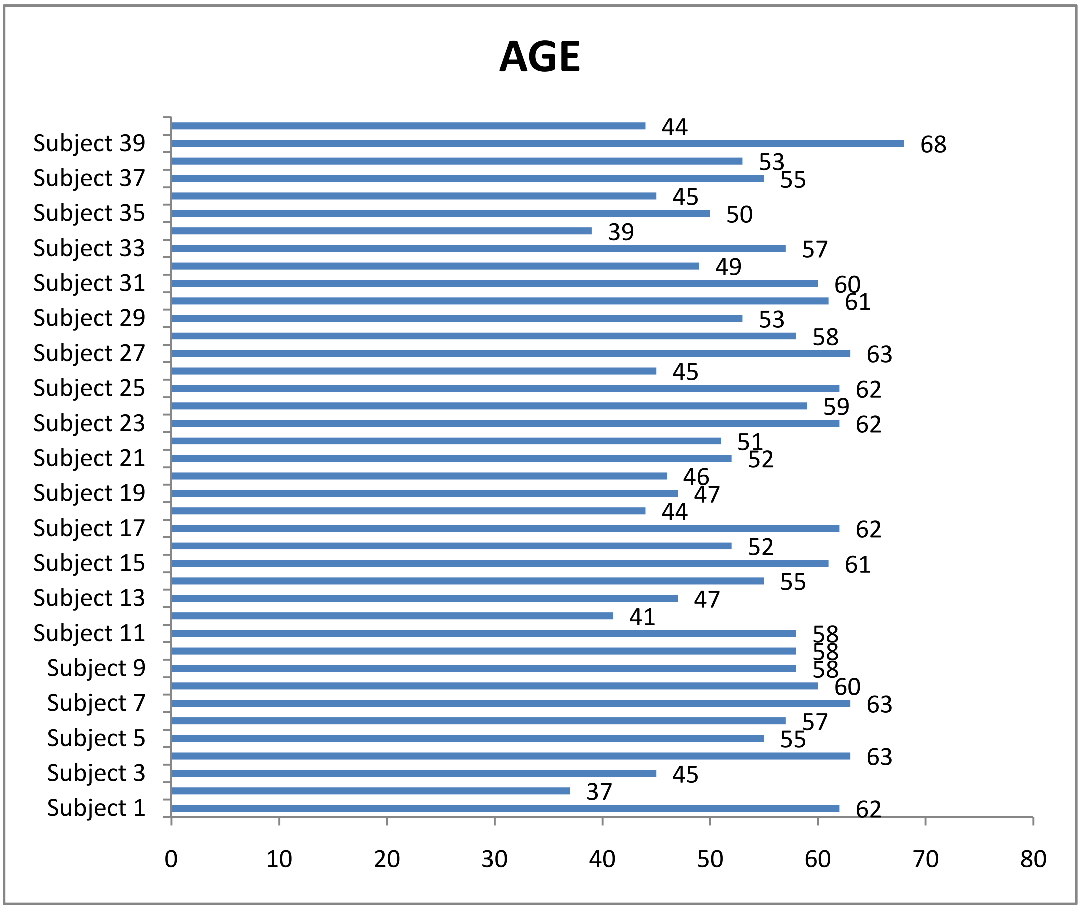
DISCUSSION

DISCUSSION

A total of forty patients suffering from Acute on Chronic Alcohol Liver disease were randomised and chosen, and divided into equal test and control arms for Betaine therapy. They were subjected to twelve weeks of therapy and weekly tests were taken to assess parameters and their pattern of change.

Age distribution: Out of forty patients, Five patients ranged from 36-44 (three in the test arm and two in the control), twelve patients between 45-54 (5 in test and 7 in control), twenty two patients between 55-64 (twelve in test arm and ten in control); and one patient aged 68 in the control arm.

CHART NO. 1 AGE



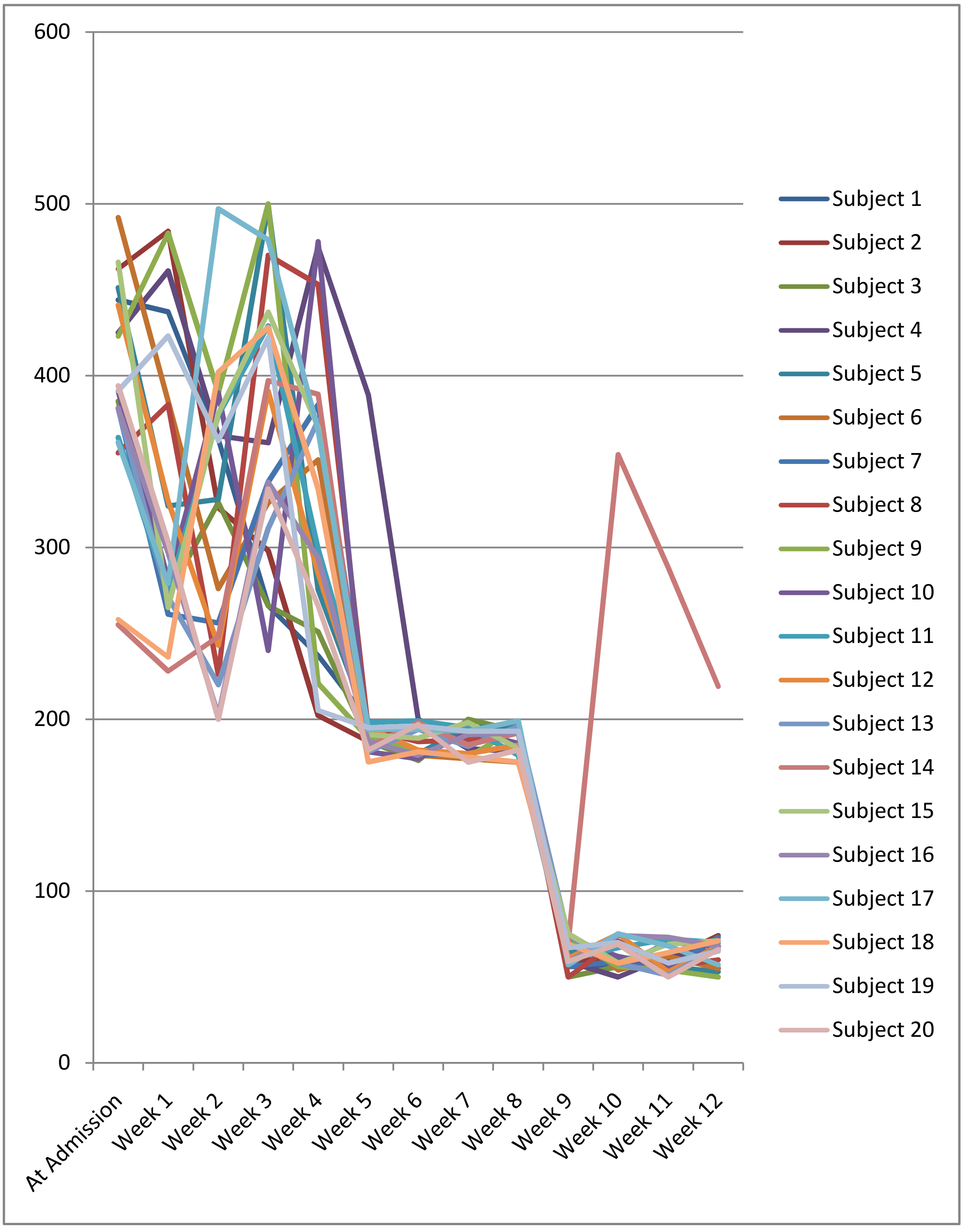
Sex:

Thirty nine males and one female made up the sample set for this study. The one female was randomly entered into the test arm. There is a definite decreased incidence of alcoholic liver disease amongst women in India due to social taboos about alcohol consumption as well as about revealing an illness related to alcohol consumption. The subject in question was well within the statistically representative realms of her group and in no was statistically significantly different from other members of her group in terms of presentation or parameters at the time of entry into study or in terms of response to her designated therapy compared to other members of her group.

Liver Function Tests:

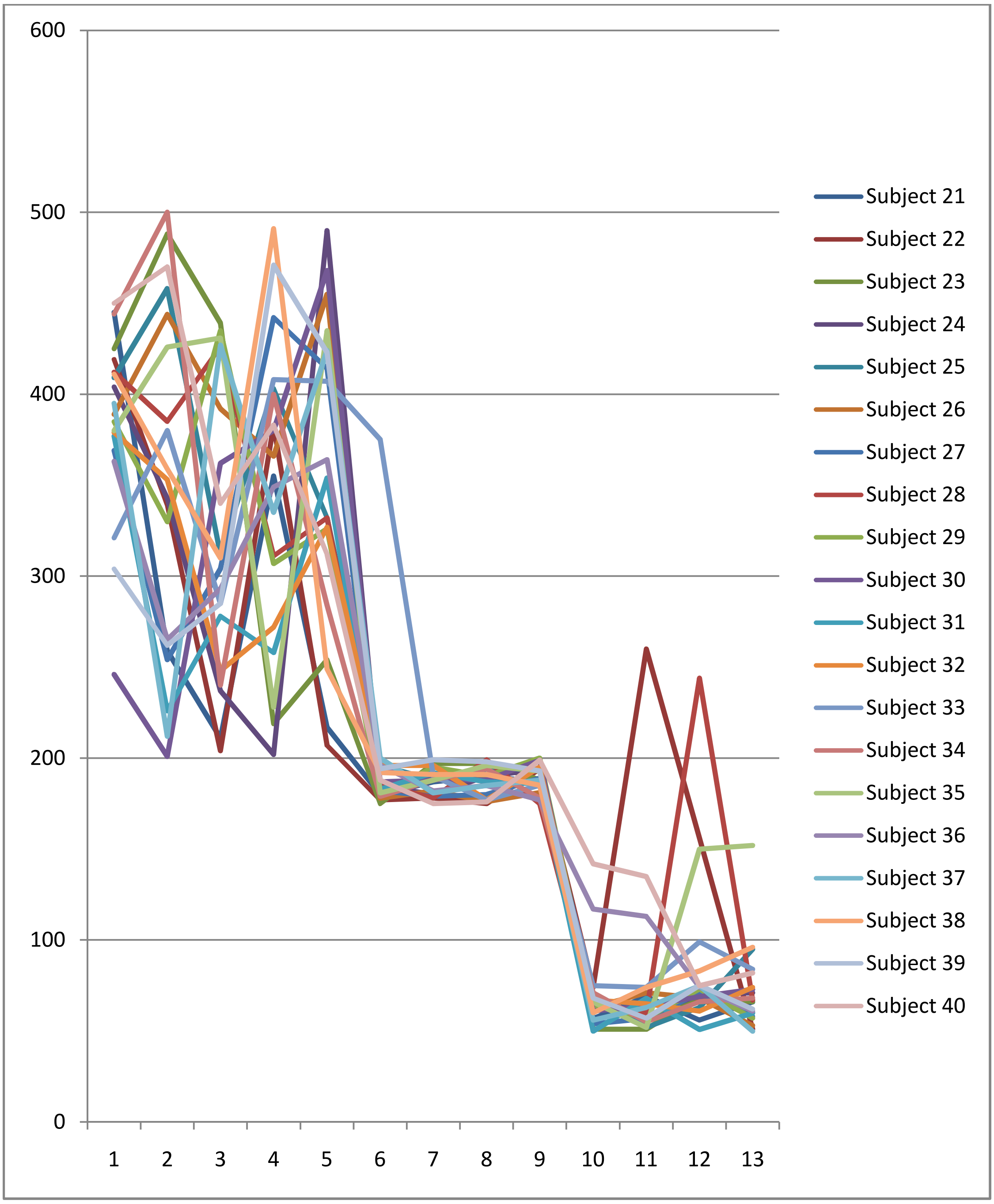
Liver function tests of all forty subjects were taken at the time of admission and every week for twelve weeks. Significant differences were found only between values of different weeks of the same patients but not between patients in the test arm and control arm. Many patients both in test and control experienced a statistically significant increase in liver enzymes and a 'rehepatitis' during the period of study in their liver enzymes. Nine Subjects (5, 6, 14, 17, 27, 33, 34, 38 and 39) experienced a rehepatitis – four in the test and five in the control arm- at 3 weeks. Seven Subjects (4, 6, 8, 9, 10, 11, 37) –six in the test, and one in control arm-experienced a rehepatitis at 4 weeks. Ten subjects (15, 17, 18, 28, 29, 30, 33, 34, 38, 39) – 3 in the test arm, seven in the control - had a rehepatitis within three weeks of study. One patient (subject 26) –control arm-had a persistent hepatitis that lasted for 4 weeks. Two patients (7,35) - one each in the test and control arm- had a rehepatitis at 5 weeks. Two patients (14,24) - one each in the test and control arm- had a rehepatitis at week 10. One patient (28) had a rehepatitis at week 11. Patterns of SGOT and SGPT were consistent with each other and no discrepancy was found between them. There was no change in alkaline phosphatase or total protein levels between different groups. Additionally, amongst the samples, there was no intersubject or interweek variation in the serum albumin levels either. Predictably the enzyme levels stayed within the realms of alcoholic hepatitis range with the SGOT zenith reaching 500. Also the bilirubin levels were statistically similar in both arms, and the increase in levels occurring expectedly just after the spike in enzyme levels. Patients only complained of a mild Gastrointestinal upset after consumption of the drug, which was comparable with placebo. No other side effects was reported. Four patients 2 in test and control each had Portal vein thrombosis as the cause for acute decompensation. The rest had continues alcohol intake as the cause.

CHART NO. 2



SGOT in test group

CHART NO. 3



SGOT in control group

CHART NO. 4

SGPT in test group

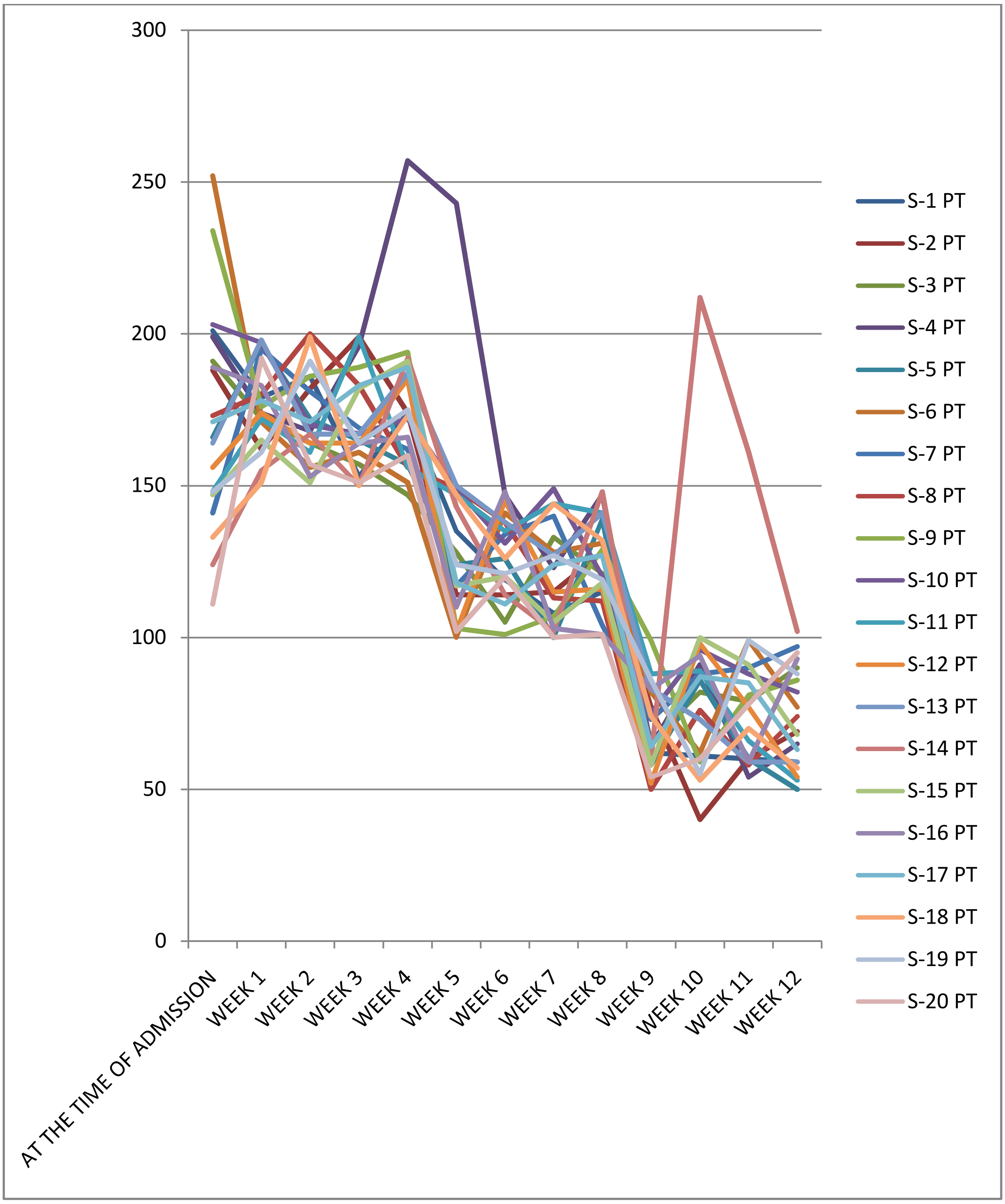
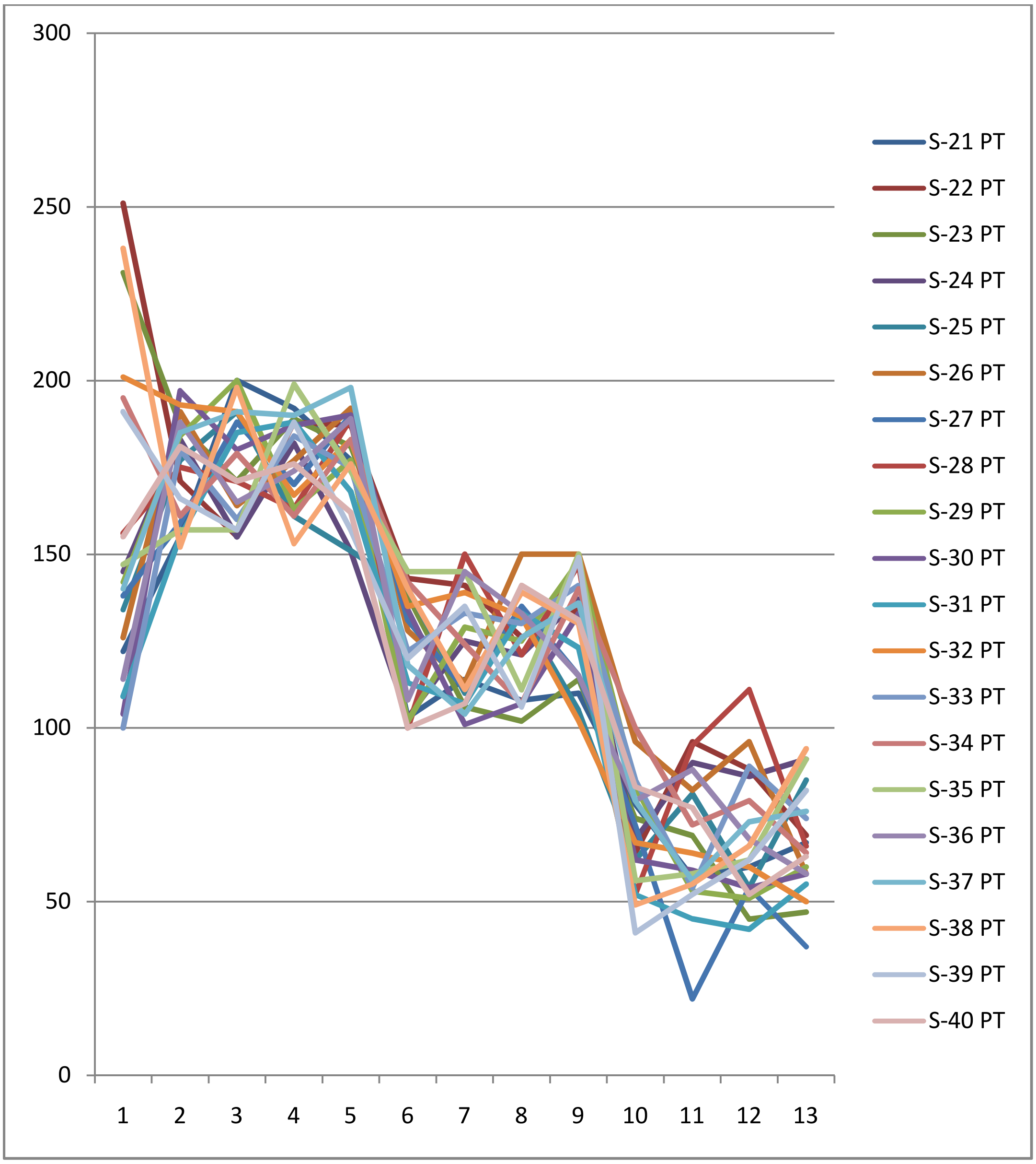


CHART NO. 5

SGPT in control group



Coagulogram:

There was no statistically significant difference in the INR alues between test and control arms indicating that the drug had no significant positive impact on the synthetic function of the inflamed liver.

Other tests:

There was no significant difference found in the renal founction test results between different groups. Many patients were mildly hyponatremic or hypokalemic probably due to volume overloaded status as the study includes only those who have objective evidence of pre-existing alcoholic chronic liver disease. Most patients also developed a mild pre-renal failure with a moderately elevated urea and only a slightly elevated creatine level concomitantly with liver decompensation and hepatitis, but recovered with recovery of liver function. Haemoglobin levels varied between different patients and between different weeks of the same patient but there was no significant difference between the two groups due to satisfactory randomization. The profile of other aspects of the complete blood count was similar. All patients remained euglycemic throughout the trial. Endoscopy results did not reveal any significant downgrading of variceal grade post therapy. Of the 20 test subjects in the test arm, 15 had grade 2 varices and 5 had grade 3, which continued to be the same post trial. In the control arm, one patient had grade 1 varices, 15 had grade 2 and 4 had grade 3; and post trial only one of them had worsened- with one subject who had grade 2 progressing to grade 3. The cause for hepatitis was all-inclusive with patients needing to have only their chronic liver disease attributable to alcohol. In the study most patients (36 in total) had the acute episode due to alcohol consumption-18 in each arm- whereas the only other cause of acute hepatitis in the sample of study was portal vein thrombosis with four patients suffering from it – two in each arm.

CONCLUSION

CONCLUSION

Betaine Therapy had no significant effect on acute on chronic alcoholic liver disease. There was no statistically significant reduction in the number of hepatitis episodes nor in the severity of the current episodes at the start of the period of study. Many patients in the test arm were found to have liver enzyme levels the same as or even more than what they were before the period of study. The drug has been tried out in two of the most common reasons for acute flares in chronic liver disease due to alcohol and yet failed to produce an impact. Thus the drug which has interested so many in the field of non alcoholic liver disease has been no better than placebo in the case of alcoholic liver disease. Since dosing has not been optimised, further studies will be required.

LIMITATIONS

LIMITATIONS

A major limitation of this study is the lack of clear optimal dose to obtain adequate efficacy of the drug. All protocols of trials involving betaine therapy use drug dosing based on the regimen used for homocystinuria. It is yet unclear if this is an over-, under- or correct dosage to test out. Another limitation is the number of patients. A larger sample set would probably be more appropriate to investigate. Thirdly, there is no correct consensus on the duration of therapy required. Perhaps a much longer trial would be required.

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BIBLIOGRAPHY

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ANNEXURES

"BETAINE THERAPY AND ITS INFLUENCE ON OUTCOME OF ACUTE ON CHRONIC ALCOHOLIC LIVER DISEASE "

PROFORMA

Name: Age/Sex:

Address: Occupation:

SYMPTOMS:

Jaundice

Altered sensorium

Upper GI bleed

Coagulopathy

Fever

PAST HISTORY:

COPD	
CVA	
CKD	
HYPOTYHROIDISM	
DIABETES MELLITUS	
VIRAL HEPATITIS	

PERSONAL HISTORY:

SMOKING

ALCOHOL

GENERAL EXAMINATION:

GCS	

VITAL SIGNS:

PR-

BP-

RR-

SYSTEMIC EXAMINATION:

CVS:

RS:

ABDOMEN:

CNS:

INVESTIGATIONS:

COMPLETE HEMOGRAM

RFT

SERUM ELECROLYTES

LFT

COAGULOGRAM

GLYCEMIC PROFILE

VIRAL HEPATITIS MARKERS

ENDOSCOPIC FINDINGS

INFORMATION SHEET

We are conducting a study on **"BETAINE THERAPY AND ITS INFLUENCE ON OUTCOME OF ACUTE ON CHRONIC ALCOHOLIC LIVER DISEASE" :** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

The purpose of this study is To study the BETAINE THERAPY AND ITS INFLUENCE ON OUTCOME OF ACUTE ON CHRONIC ALCOHOLIC LIVER DISEASE

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

The process of dividing patients who are selected will be random and it shall not be determined prior or during study, which group the patient is on. The sanctity of single blinding shall be maintained. The patient may be ascribed to the placebo group whereby he or she may be denied any benefit or harm the drug may or may not induce.

The patient must wilfully accept any known side effects of the drug if he or she is taking so.

Signature of Investigator   Signature of Participant

Date :
Place :

PATIENT CONSENT FORM

Study Detail : “**BETAINE THERAPY AND ITS INFLUENCE ON OUTCOME OF ACUTE ON CHRONIC ALCOHOLIC LIVER DISEASE** ”

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Patient’s Name :

Patient’s Age :

Identification Number :

Patient may check (√) these boxes

a) I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

b) I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

c) I understand that sponsor of the clinical study, others working on the sponsor’s behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

d) I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

e) I hereby consent to participate in this study. ☐

f) I hereby give permission to undergo detailed clinical examination and blood investigations as required. ☐

Signature/thumb impression

Patient’s Name and Address: Signature of Investigator

Study Investigator’s Name:
Dr. VENKATAKRISHNAN R.

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No. 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.Venkatakrishnan
Postgraduate M.D.(General Medicine)
Madras Medical College
Chennai 600 003

Dear Dr.Venkatakrishnan,

The Institutional Ethics Committee has considered your request and approved your study titled **"Betaine therapy and its effects on acute on chronic alcoholic liver disease" No.16072015.**

The following members of Ethics Committee were present in the meeting held on 07.07.2015 conducted at Madras Medical College, Chennai-3.

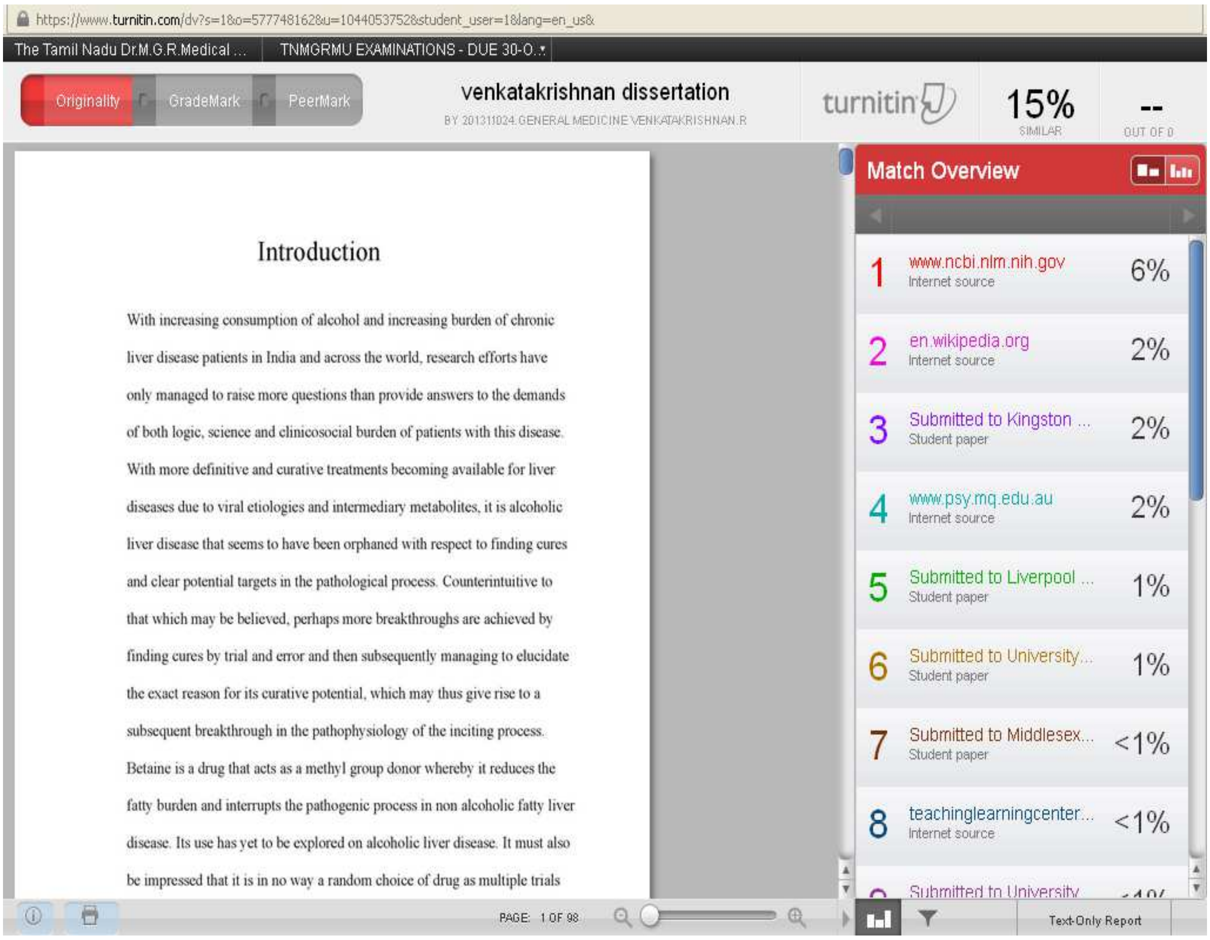
1. Prof.C.Rajendran, M.D.,	: Chairperson
2. Prof.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
3. Prof.Sudha Seshayyan, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4. Prof.B.Vasanthi, M.D., Professor Pharmacology, MMC	: Member
5. Prof.P.Ragumani, M.S., Professor, Inst.of Surgery, MMC	: Member
6. Prof.Md.Ali, M.D., D.M., Prof. & HOD of Medi.G.E., MMC	: Member
7. Prof.Baby Vasumathi, Director, Inst.of O&G, Ch-8	: Member
8. Prof.K.Ramadevi, Director, Inst.of Biochemistry, MMC	: Member
9. Prof.Saraswathy, M.D., Director, Inst. Of Pathology, MMC	: Member
10. Prof.Srinivasagalu, Director, Inst.of Inter Med. MMC	: Member
11. Thiru S.Rameshkumar, B.Com., MBA	: Lay Person
12. Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
13. Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progres of the study and SAE occurring in the course of the study, any changes in th protocol and patients information/informed consent and asks to be provided a co of the final report.

Member Secretary, Ethics Commi

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003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Introduction
With increasing consumption of alcohol and increasing burden of chronic liver disease patients in India and across the world, research efforts have only managed to raise more questions than provide answers to the demands of both logic, science and clinicosocial burden of patients with this disease. With more definitive and curative treatments becoming available for liver diseases due to viral etiologies and intermediary metabolites, it is alcoholic liver disease that seems to have been orphaned with respect to finding cures and clear potential targets in the pathological process. Counterintuitive to that which may be believed, perhaps more breakthroughs are achieved by finding cures by trial and error and then subsequently managing to elucidate the exact reason for its curative potential, which may thus give rise to a subsequent breakthrough in the pathophysiology of the inciting process. Betaine is a drug that acts as a methyl group donor whereby it reduces the fatty burden and interrupts the pathogenic process in non alcoholic fatty liver disease. Its use has yet to be explored on alcoholic liver disease. It must also be impressed that it is in no way a random choice of drug as multiple trials
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Introduction

MASTER CHART

PATIENT	Parameters	ADMISSION	WEEK 1	WEEK 2	WEEK 3	WEEK 4	WEEK 5	WEEK 6	WEEK 7	WEEK 8	WEEK 9	WEEK 10	WEEK 11	WEEK 12
	AGE	62	62	62	62	62	62	62	62	62	62	62	62	62
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	46000	48000	50000	55000	46000	40000	41000	56000	47000	42000	48000	51000	54000
	HB	8.7	8.6	8.1	8.4	8.2	8.7	8	7.9	8.4	7.6	7.7	7.3	7.5
	PCV	26	25	26	21	24	25	22	23	27	22	22	20	25
	TC (in thousands)	7.3	10.3	7.4	6.3	11.0	5.2	7.5	5.3	10.5	5.0	10.4	10.0	8.8
	UREA	56	48	44	37	36	39	41	49	45	26	43	31	36
	CREATINE	1.5	1.2	1.1	1.5	1.4	0.9	0.7	0.8	0.6	1.2	1.6	1.1	0.9
	SODIUM	128	130	130	126	128	122	136	127	131	129	133	135	129
	POTASSIUM	2.4	2.6	3.2	3.1	3.7	3.5	3	3.5	3.5	3.5	2.9	3.6	3.1
	T.BILIRUBIN	11	12.0	12.9	11.0	11.1	6.2	7.7	7.9	5.7	2.2	1.9	2.3	3.2
SUBJECT 1	D.BILIRUBIN	1.9	2.9	3.7	4.0	2.1	3.6	2.5	3.5	3.5	1.2	1.5	1.1	2.2
	OT	444	437	363	266	237	199	178	194	185	56	56	70	68
	PT	201	179	186	153	174	135	119	108	115	62	61	60	59
	ALP	54	69	70	58	68	62	63	58	54	53	69	65	59
	T.PROTEIN	5.5	5.4	5.0	5.4	5.5	5.3	5.5	4.9	5.2	5.4	5.0	5.2	5.6
	S.ALBUMIN	2,7	2.6	2.7	2.6	2.8	2.7	2.8	2.4	2.5	2.7	2.6	2.7	2.8
	INR	3.3	3.3	3.2	2.3	1.7	2.2	2.0	1.9	1.8	1.3	1.4	1.3	1.4
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	Normal	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 3 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	37	37	37	37	37	37	37	37	37	37	37	37	37
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	52000	51000	53000	58000	49000	43000	44000	59000	50000	45000	51000	54000	57000
	HB	8.9	9.1	8.4	8.7	8.5	9	8.3	8.2	8.7	7.9	8	7.3	7.5
	PCV	26	27	25	23	21	25	21	23	24	26	21	24	20
	TC (in thousands)	5.9	4.7	5.4	10.3	7.1	9.9	6.2	8.2	7.3	8.2	6.4	5.0	10.9
	UREA	61	53	49	42	41	44	46	54	50	31	48	36	41
	CREATINE	1.4	0.9	0.8	1.2	1.6	0.6	0.4	0.5	0.3	0.9	1.3	0.8	0.9
	SODIUM	134	131	131	127	129	123	137	128	132	130	134	136	129
	POTASSIUM	4.2	3	3.6	3.5	4.1	3.9	3.4	3.9	3.9	3.9	3.3	4	3.5
	T.BILIRUBIN	14.6	12.2	11.5	12.7	11.7	7.3	5.9	6.3	7.8	3.8	2.1	2.1	3.0
SUBJECT 2	D.BILIRUBIN	3.5	2.7	3.6	3.9	4.0	3.4	3.9	3.1	2.5	1.7	1.1	0.6	2.0
	OT	462	484	323	298	202	187	196	193	195	59	61	60	74
	PT	188	162	182	199	174	114	114	115	128	77	40	60	69
	ALP	66	50	65	50	67	70	62	66	70	59	61	50	59
	T.PROTEIN	5.2	5.0	5.5	5.3	5.6	5.5	5.3	5.0	4.8	5.0	4.8	5.5	5.2
	S.ALBUMIN	2.6	2.4	2.6	2.7	2.5	2.6	2.4	2.5	2.6	2.5	2.5	2.6	2.5
	INR	4.1	3.3	3.3	1.9	1.6	2.1	1.6	2.1	2.4	1.8	2.0	2.2	2.4
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	45	45	45	45	45	45	45	45	45	45	45	45	45
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	51000	47000	49000	54000	45000	39000	40000	55000	46000	41000	47000	50000	53000
	HB	7.6	7.8	8.1	8.4	8.2	8.7	8	7.9	8.4	7.6	7.7	7.3	7.5
	PCV	24	22	24	22	24	22	22	26	25	23	24	25	27
	TC (in thousands)	9.9	8.2	8.5	6.9	7.7	9.4	6.3	8.9	7.9	7.9	10.0	11.0	7.4
	UREA	44	33	53	33	35	33	33	40	39	40	33	42	38
	CREATINE	1.2	1.1	1.4	1.4	1.3	1.5	1.1	1.0	1.3	1.2	1.2	1.1	1.5
	SODIUM	135	131	136	131	134	129	131	131	130	130	132	134	133
	POTASSIUM	3.2	4.4	4.8	2.8	4.4	4.7	3.4	2.5	2.7	4.0	4.0	3.1	3.0
	T.BILIRUBIN	11.3	13.4	11.8	11.5	10.0	6.6	7.1	7.8	5.1	3.1	2.6	3.3	3.3
SUBJECT 3	D.BILIRUBIN	4.1	3.1	2.9	3.9	2.1	3.1	3.8	2.8	2.7	1.4	0.7	2.0	2.0
	OT	385	272	326	266	251	187	176	200	193	50	56	62	55
	PT	191	172	164	157	147	128	105	133	121	66	82	79	90
	ALP	60	70	50	63	57	63	57	70	66	60	54	57	51
	T.PROTEIN	5.4	5.2	5.5	4.9	5.3	5.6	5.1	5.2	4.8	5.5	5.0	4.8	5.4
	S.ALBUMIN	2.6	2.7	2.7	2.5	2.8	2.8	2.6	2.7	2.7	2.7	2.4	2.6	2.5
	INR	3,4	3.1	3.8	1.6	1.6	2.1	1.7	1.9	2.3	2.1	2.0	2.2	2.0
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE

	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	63	63	63	63	63	63	63	63	63	63	63	63	63
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	55000	53000	55000	60000	51000	45000	46000	61000	52000	47000	53000	56000	59000
	HB	8.1	7.7	7.2	7.5	7.3	7.8	7.1	7	7.5	6.7	6.8	6.4	6.6
	PCV	25	20	24	25	20	24	21	23	26	24	22	25	20
	TC (in thousands)	10.1	5.8	8.5	10.7	4.2	6.1	10.6	7.5	7.8	7.4	7.3	10.8	9.8
	UREA	55	37	31	42	36	54	49	52	44	30	34	34	30
	CREATINE	1.6	1.3	1.1	1.4	1.4	1.0	1.1	1.2	1.4	1.4	1.0	1.0	1.4
	SODIUM	128	132	128	127	135	132	135	128	129	127	134	137	129
	POTASSIUM	2.6	4.1	2.8	3.8	3.6	4.1	4.1	3.9	3.7	3.6	4.0	4.1	2.4
	T.BILIRUBIN	9.1	12.5	13.5	11.7	13.5	18.1	13.4	8.6	5.1	3.0	3.0	2.4	3.5
SUBJECT 4	D.BILIRUBIN	2.6	3.9	3.4	3.4	3.9	10.0	3.4	3.3	2.4	1.6	1.8	0.7	1.4
	OT	425	461	365	361	475	389	200	183	181	59	50	62	66
	PT	199	174	168	196	257	243	147	123	147	64	91	54	65
	ALP	64	61	61	64	55	57	70	50	52	51	57	70	55
	T.PROTEIN	5.1	5.0	5.2	5.1	5.6	4.8	5.6	5.6	4.8	5.0	5.0	5.2	5.0
	S.ALBUMIN	2.4	2.5	2.6	2.5	2.6	2.6	2.7	2.7	2.8	2.7	2.5	2.6	2.7
	INR	4.5	4.0	3.4	1.5	3.9	3.1	2.2	1.6	2.0	1.6	2.3	2.4	1.9
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis
	AGE	55	55	55	55	55	55	55	55	55	55	55	55	55
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	52000	57000	59000	64000	55000	49000	50000	65000	56000	51000	57000	60000	63000
	HB	8.5	8.8	8.3	8.6	8.4	8.9	8.2	8.1	8.6	7.8	7.9	7.5	7.7
	PCV	26	27	22	25	21	25	22	24	23	22	24	26	21
	TC (in thousands)	9.2	8.6	9.4	5.8	7.1	4.6	6.9	6.1	7.9	6.0	5.3	9.6	9.1
	UREA	58	41	48	36	33	46	48	52	39	45	32	47	33
	CREATINE	1.4	1.3	1.1	1.1	1.1	1.2	1.2	1.1	1.1	1.1	1.3	1.4	1.2
	SODIUM	132	136	135	135	130	134	128	131	133	129	131	133	132
	POTASSIUM	2.8	4.3	4.5	2.7	2.5	3.6	4.2	4.6	3.3	4.4	3.8	2.6	3.3
	T.BILIRUBIN	15.1	11.6	11.5	11.0	13.9	5.5	5.7	6.1	6.7	2.1	3.9	3.8	3.4
SUBJECT 5	D.BILIRUBIN	4.1	3.8	3.7	3.4	2.6	4.0	3.6	2.9	3.2	0.2	1.0	1.3	1.3
	OT	451	324	328	499	275	197	180	195	196	65	62	56	53
	PT	166	197	172	165	157	124	126	100	139	58	86	60	50
	ALP	60	63	57	52	58	63	59	59	53	63	58	56	52
	T.PROTEIN	5.6	5.5	5.3	5.2	4.8	4.9	5.3	5.0	5.0	5.0	5.5	5.3	5.1
	S.ALBUMIN	2.4	2.5	2.5	2.7	2.5	2.6	2.6	2.5	2.4	2.5	2.6	2.6	2.5
	INR	4.9	3.1	3.2	1.8	1.7	1.6	2.4	2.0	1.6	1.9	1.8	2.0	1.7
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	57	57	57	57	57	57	57	57	57	57	57	57	57
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	46000	42000	44000	49000	40000	34000	35000	50000	41000	36000	42000	45000	48000
	HB	8.4	8.2	7.7	8	7.8	8.3	7.6	7.5	8	7.2	7.3	6.9	7.1
	PCV	25	27	25	25	21	21	25	23	21	24	22	19	27
	TC (in thousands)	13.1	5.2	9.0	7.6	8.8	5.5	6.1	4.4	10.7	6.7	8.5	5.2	5.3
	UREA	84	35	39	36	50	37	39	50	41	49	43	51	34
	CREATINE	1.1	1.3	1.4	1.0	1.3	1.4	1.3	1.0	1.3	1.2	1.3	1.3	1.1
	SODIUM	130	129	135	133	129	132	136	127	134	128	130	128	127
	POTASSIUM	3.2	3.0	3.5	3.4	4.7	3.4	2.8	3.5	3.8	3.2	3.7	3.0	3.2
	T.BILIRUBIN	18.2	11.7	11.8	13.6	13.2	6.9	6.7	7.5	6.8	3.9	2.5	3.4	2.7
SUBJECT 6	D.BILIRUBIN	4.4	2.9	3.2	3.7	2.9	3.2	2.4	2.1	2.4	2.9	1.9	0.8	1.1
	OT	492	385	276	326	351	193	179	177	175	75	54	62	55
	PT	252	171	156	161	151	100	141	128	131	82	62	99	77
	ALP	80	54	52	51	53	61	58	57	67	55	70	69	58
	T.PROTEIN	5.9	5.0	5.4	5.1	5.6	5.2	5.1	5.1	5.3	5.4	4.9	5.4	5.1

	S.ALBUMIN	2.4	2.5	2.7	2.6	2.8	2.8	2.4	2.6	2.4	2.5	2.5	2.6	2.7
	INR	4.8	3.9	3.1	1.9	2.4	2.1	1.6	2.4	2.4	1.6	2.2	1.7	2.0
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	63	63	63	63	63	63	63	63	63	63	63	63	63
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	55000	45000	47000	52000	43000	37000	38000	53000	44000	39000	45000	48000	51000
	HB	8.3	8	7.5	7.8	7.6	8.1	7.4	7.3	7.8	7	7.1	6.7	6.9
	PCV	29	20	24	24	24	23	27	26	24	21	20	25	24
	TC (in thousands)	16.1	7.9	9.6	7.9	7.4	6.4	11.0	5.8	9.8	9.9	4.2	6.4	6.5
	UREA	51	55	39	36	33	39	42	47	45	44	31	42	49
	CREATINE	1.8	1.5	1.1	1.4	1.4	1.4	1.3	1.3	1.5	1.4	1.4	1.0	1.3
	SODIUM	126	130	131	127	131	136	135	137	136	132	135	128	136
	POTASSIUM	4.4	2.7	3.1	2.5	3.0	3.1	3.7	2.9	3.7	2.7	3.0	3.7	2.4
	T.BILIRUBIN	14.4	12.9	12.3	13.1	13.7	7.6	7.5	7.4	7.4	3.1	3.1	2.1	3.8
SUBJECT 7	D.BILIRUBIN	4.6	3.4	2.6	2.2	3.4	3.9	3.6	3.0	2.9	0.9	1.7	0.4	2.5
	OT	381	261	256	338	383	193	179	180	185	56	59	55	73
	PT	141	195	181	169	162	117	134	140	104	73	88	90	97
	ALP	68	62	66	66	52	55	59	68	60	70	62	54	68
	T.PROTEIN	5.9	5.6	5.0	5.4	5.2	5.2	5.5	5.3	5.2	5.5	5.6	4.8	5.4
	S.ALBUMIN	2.6	2.5	2.6	2.6	2.5	2.5	2.6	2.6	2.5	2.7	2.5	2.6	2.4
	INR	3.8	3.5	4.1	1.9	2.2	2.0	2.0	2.3	1.7	1.9	2.1	1.8	1.8
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	60	60	60	60	60	60	60	60	60	60	60	60	60
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	58000	51000	53000	58000	49000	43000	44000	59000	50000	45000	51000	54000	57000
	HB	8.7	8.9	9.1	7.7	7.9	8.9	7.9	7.9	8.1	8.4	8.3	8.9	8.9
	PCV	26	25	25	20	19	27	21	21	25	24	24	29	23
	TC (in thousands)	11.4	9.2	6.5	5.3	9.9	10.2	4.5	7.9	8.4	6.8	11.0	10.2	5.2
	UREA	62	35	52	30	53	45	35	44	38	52	41	54	37
	CREATINE	1.5	1.4	1.5	1.3	1.5	1.4	1.0	1.3	1.2	1.2	1.4	1.4	1.2
	SODIUM	132	131	134	131	135	127	128	136	133	130	135	131	132
	POTASSIUM	3.6	3.1	3.1	4.6	4.0	3.9	3.8	4.1	3.7	2.4	3.2	3.3	3.6
	T.BILIRUBIN	10.4	12.0	13.0	13.6	11.9	12.2	14.1	9.7	5.2	3.3	3.3	2.6	3.1
SUBJECT 8	D.BILIRUBIN	2.9	3.8	2.4	2.5	3.7	3.6	3.6	2.4	3.8	1.3	1.1	1.3	1.1
	OT	355	383	226	470	453	194	187	188	192	50	71	56	60
	PT	173	180	200	183	156	149	138	113	112	50	76	58	74
	ALP	50	59	52	66	65	65	68	61	50	62	69	50	52
	T.PROTEIN	5.2	5.3	5.1	5.4	4.9	5.2	5.2	5.3	5.5	5.2	5.6	5.3	4.8
	S.ALBUMIN	2.8	2.6	2.5	2.5	2.5	2.7	2.7	2.5	2.5	2.7	2.5	2.6	2.6
	INR	4.3	3.8	3.7	1.8	2.0	1.9	1.8	2.0	1.9	2.2	2.3	1.8	2.2
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	58	58	58	58	58	58	58	58	58	58	58	58	58
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	33000	38000	40000	45000	36000	30000	31000	46000	37000	32000	38000	41000	44000
	HB	10	9.9	8.1	8.4	8.2	8.7	8	7.9	8.4	7.6	7.7	7.3	7.5
	PCV	34	27	26	25	25	24	21	19	24	18	25	19	24
	TC (in thousands)	3.7	4.9	10.9	6.4	10.9	7.6	8.4	4.6	9.9	5.3	6.5	10.5	5.5
	UREA	77	32	40	50	32	37	47	45	32	38	38	31	39
	CREATINE	1.2	1.1	1.0	1.3	1.5	1.0	1.2	1.5	1.4	1.0	1.1	1.1	1.2
	SODIUM	146	131	128	137	136	131	129	129	137	134	131	136	137
	POTASSIUM	2.4	4.4	3.3	3.3	4.2	3.8	2.6	4.4	4.4	2.7	3.2	4.7	4.0
	T.BILIRUBIN	15.1	11.9	11.3	12.7	11.4	8.5	9.3	5.9	5.6	2.4	2.9	2.9	3.7
SUBJECT 9	D.BILIRUBIN	6.1	3.8	2.3	3.3	3.3	2.9	3.4	3.7	2.4	1.6	1.3	1.7	2.0
	OT	423	483	390	500	221	189	182	178	195	73	56	54	50

	PT	234	176	186	189	194	103	101	107	129	99	59	81	86
	ALP	54	68	67	54	70	51	57	59	51	62	58	52	53
	T.PROTEIN	5.2	5.1	5.1	5.2	5.5	4.9	5.4	4.8	5.3	5.1	5.5	5.0	5.2
	S.ALBUMIN	2.5	2.6	2.5	2.5	2.6	2.4	2.7	2.7	2.5	2.5	2.6	2.6	2.5
	INR	4.1	3.8	3.5	2.3	2.1	2.1	1.6	1.6	2.3	1.6	2.4	2.5	1.7
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	58	58	58	58	58	58	58	58	58	58	58	58	58
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	59000	54000	56000	61000	52000	46000	47000	62000	53000	48000	54000	57000	60000
	HB	8.3	8.9	8.4	8.7	8.5	9	8.3	8.2	8.7	7.9	8	7.6	7.8
	PCV	26	25	26	26	23	26	19	19	21	26	25	21	20
	TC (in thousands)	10.4	7.2	7.3	9.1	8.1	10.3	8.8	5.5	5.3	7.5	7.5	7.2	8.1
	UREA	41	54	49	42	31	31	52	31	35	45	44	33	37
	CREATINE	1.1	1.2	1.5	1.0	1.1	1.3	1.3	1.2	1.3	1.3	1.2	1.0	1.3
	SODIUM	127	129	130	132	133	129	131	132	131	135	134	135	127
	POTASSIUM	3.8	2.6	2.6	2.7	3.3	3.8	3.6	2.5	3.6	4.4	4.2	4.3	3.5
	T.BILIRUBIN	16.4	12.7	12.7	12.1	12.1	15.1	5.9	7.5	7.1	2.4	2.8	2.5	2.6
SUBJECT 10	D.BILIRUBIN	6.8	3.1	3.1	2.4	2.0	2.2	3.8	3.5	3.4	1.2	1.8	1.5	1.5
	OT	391	281	390	240	478	181	177	193	186	73	62	55	68
	PT	203	197	170	167	172	149	131	149	120	75	96	88	82
	ALP	74	70	60	55	53	56	63	52	50	58	63	68	57
	T.PROTEIN	5.5	5.4	5.1	5.4	4.8	4.8	5.0	5.3	4.9	5.3	4.9	5.3	5.5
	S.ALBUMIN	2.6	2.6	2.6	2.6	2.8	2.6	2.7	2.8	2.7	2.6	2.4	2.7	2.7
	INR	4.4	3.8	3.0	2.1	4.2	2.2	1.7	2.4	1.9	2.5	1.9	2.2	2.4
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	58	58	58	58	58	58	58	58	58	58	58	58	58
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	51000	45000	47000	52000	43000	37000	38000	53000	44000	39000	45000	48000	51000
	HB	10.1	10	9.5	9.8	9.6	10.1	9.4	9.3	9.8	9	9.1	8.7	8.9
	PCV	32	24	24	27	26	30	25	28	26	23	21	22	23
	TC (in thousands)	15.7	7.8	4.6	6.5	9.3	9.1	4.1	6.0	6.2	10.5	7.7	8.5	7.5
	UREA	41	55	55	46	40	52	48	31	39	42	42	54	31
	CREATINE	0.6	1.1	1.1	1.4	1.0	1.2	1.4	1.5	1.4	1.2	1.5	1.1	1.5
	SODIUM	144	137	135	129	134	135	137	132	128	133	129	130	137
	POTASSIUM	3.8	3.5	4.0	2.9	4.0	3.2	3.8	4.3	4.8	4.3	2.8	3.5	4.6
	T.BILIRUBIN	15.1	13.8	13.1	13.3	12.3	7.7	5.3	5.9	6.3	2.4	3.9	3.0	2.3
SUBJECT 11	D.BILIRUBIN	4.3	3.0	2.1	2.8	3.6	3.1	3.5	2.8	3.0	1.4	2.5	1.6	1.1
	OT	364	273	377	429	299	198	199	195	179	60	67	72	70
	PT	148	172	161	199	156	147	135	144	141	88	89	66	53
	ALP	50	59	69	62	66	66	58	66	50	57	68	50	60
	T.PROTEIN	5.7	5.1	5.0	5.5	5.0	5.3	5.0	5.1	5.5	5.0	5.4	5.1	5.5
	S.ALBUMIN	2.4	2.7	2.4	2.7	2.5	2.6	2.6	2.6	2.5	2.7	2.6	2.8	2.7
	INR	4.5	4.1	3.7	2.0	4.4	2.0	1.6	2.5	2.0	2.2	1.6	2.4	2.1
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	41	41	41	41	41	41	41	41	41	41	41	41	41
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	30000	34000	36000	41000	32000	26000	27000	42000	33000	28000	34000	37000	40000
	HB	7.5	8	7.5	7.8	7.6	8.1	7.4	7.3	7.8	7	7.1	6.7	6.9
	PCV	24	27	26	18	21	21	27	21	25	22	20	26	18
	TC (in thousands)	16.8	10.2	9.4	8.4	6.3	9.4	8.8	8.2	10.4	9.5	7.5	5.8	10.8
	UREA	61	37	41	47	41	53	30	45	38	39	40	34	33
	CREATINE	1.3	1.0	1.4	1.0	1.3	1.2	1.2	1.4	1.3	1.5	1.3	1.3	1.5
	SODIUM	142	129	137	132	127	131	133	128	136	127	134	127	131
	POTASSIUM	4.4	3.3	2.6	3.9	3.2	2.8	4.5	4.4	4.2	4.1	3.3	3.4	3.2

	T.BILIRUBIN	13.5	12.8	13.4	12.9	11.7	7.7	5.0	6.3	6.7	3.2	3.2	2.8	2.5
SUBJECT 12	D.BILIRUBIN	4.8	2.3	3.9	2.4	2.8	3.3	3.1	2.9	2.0	1.5	1.6	1.2	1.0
	OT	441	327	243	391	285	195	182	180	185	61	75	53	68
	PT	156	174	164	164	185	102	145	115	116	52	98	77	54
	ALP	62	55	57	56	54	66	56	61	64	65	60	64	55
	T.PROTEIN	5.3	5.0	5.1	5.4	5.3	5.5	5.5	5.5	5.5	5.3	5.4	5.5	5.4
	S.ALBUMIN	2.5	2.7	2.7	2.5	2.5	2.6	2.7	2.7	2.7	2.4	2.5	2.4	2.4
	INR	3.9	4.2	3.3	1.7	2.2	2.3	1.8	2.1	2.2	2.3	1.8	2.0	2.3
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 3 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	47	47	47	47	47	47	47	47	47	47	47	47	47
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	68000	70000	72000	77000	68000	62000	63000	78000	69000	64000	70000	73000	76000
	HB	7.4	7.1	6.6	6.9	6.7	7.2	6.5	6.4	6.9	6.1	6.2	5.8	6
	PCV	26	24	19	23	21	26	26	26	22	18	20	25	23
	TC (in thousands)	4.1	5.5	5.3	4.8	8.8	7.3	10.4	9.4	8.1	4.1	8.4	5.8	8.3
	UREA	39	42	55	45	31	41	45	55	35	53	39	53	47
	CREATINE	0.7	1.2	1.4	1.5	1.1	1.4	1.2	1.4	1.2	1.2	1.5	1.3	1.2
	SODIUM	135	134	134	128	128	128	135	132	133	136	133	128	130
	POTASSIUM	4	3.7	3.5	4.8	2.9	4.6	4.5	4.5	3.7	3.9	2.7	2.5	3.5
	T.BILIRUBIN	16.1	13.7	12.5	11.2	11.4	5.5	7.4	6.8	6.1	3.9	3.4	3.8	3.8
SUBJECT 13	D.BILIRUBIN	7.1	3.5	2.3	3.7	3.7	3.0	3.3	3.5	2.2	2.6	2.1	2.8	1.8
	OT	381	271	220	311	374	181	194	184	196	74	58	51	67
	PT	164	198	167	167	187	150	138	127	141	83	73	59	59
	ALP	64	62	69	70	68	59	63	62	54	68	65	53	51
	T.PROTEIN	5.5	5.0	5.4	4.9	5.3	5.0	5.0	5.4	4.9	5.4	5.2	5.0	5.4
	S.ALBUMIN	2.9	2.5	2.8	2.8	2.6	2.5	2.7	2.8	2.5	2.6	2.7	2.6	2.8
	INR	3.9	3.8	3.7	2.5	2.3	1.5	1.8	1.5	1.7	1.6	1.9	2.1	2.3
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	55	55	55	55	55	55	55	55	55	55	55	55	55
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	66000	66000	68000	73000	64000	58000	59000	74000	65000	60000	66000	69000	72000
	HB	6	6.1	5.6	5.9	5.7	6.2	5.5	5.4	5.9	5.1	5.2	4.8	5
	PCV	21	25	21	19	21	23	19	27	25	24	26	24	27
	TC (in thousands)	13.2	9.4	7.5	4.6	10.6	4.3	10.1	7.7	5.1	7.5	7.1	10.8	5.2
	UREA	41	54	54	38	47	44	44	32	55	36	39	44	53
	CREATINE	1.2	1.1	1.4	1.3	1.1	1.3	1.4	1.4	1.4	1.4	1.1	1.3	1.2
	SODIUM	131	127	133	127	128	136	133	129	132	137	137	132	133
	POTASSIUM	4.1	2.6	4.6	3.0	3.1	3.6	2.7	4.3	4.3	3.1	3.5	3.7	3.2
	T.BILIRUBIN	15.2	12.5	12.5	11.4	12.6	11.6	9.0	5.2	7.4	5.6	4.6	11.4	10.1
SUBJECT 14	D.BILIRUBIN	6.3	2.5	3.6	3.5	2.7	3.3	3.8	2.3	3.5	3.6	2.5	4.6	3.3
	OT	255	228	248	397	389	184	198	185	192	70	354	288	219
	PT	124	155	167	150	192	143	114	102	148	60	212	161	102
	ALP	90	69	58	64	63	64	70	69	66	70	66	51	70
	T.PROTEIN	5.7	4.9	4.9	5.2	5.5	5.4	5.0	5.2	5.6	5.1	5.3	5.2	5.0
	S.ALBUMIN	2.8	2.7	2.6	2.6	2.7	2.8	2.5	2.6	2.4	2.6	2.8	2.7	2.4
	INR	4.5	3.8	4.1	1.8	1.5	2.5	1.7	2.3	2.2	1.9	2.1	1.5	2.1
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	61	61	61	61	61	61	61	61	61	61	61	61	61
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	44000	40000	42000	47000	38000	32000	33000	48000	39000	34000	40000	43000	46000
	HB	9.1	9.9	9.4	9.7	9.5	10	9.3	9.2	9.7	8.9	9	8.6	8.8
	PCV	26	28	24	25	21	22	26	25	24	23	19	18	25
	TC (in thousands)	11	8.6	4.6	7.8	8.1	8.4	5.0	10.0	8.5	4.2	10.4	4.2	10.1
	UREA	61	55	55	49	33	31	44	32	30	31	53	53	33

	CREATINE	1.3	1.1	1.0	1.2	1.1	1.0	1.2	1.4	1.0	1.2	1.3	1.5	1.2
	SODIUM	137	134	129	134	134	134	129	128	134	135	127	137	137
	POTASSIUM	4.6	3.2	2.6	3.9	4.1	2.9	3.2	2.8	3.0	2.8	3.3	2.7	4.4
	T.BILIRUBIN	14.9	12.9	12.1	12.5	13.8	7.4	5.0	7.8	6.5	3.6	3.6	3.3	2.9
SUBJECT 15	D.BILIRUBIN	3.5	2.2	3.1	2.2	2.0	2.1	3.7	2.2	2.5	1.5	1.7	1.2	1.4
	OT	466	265	378	437	370	191	189	198	183	75	58	69	71
	PT	147	165	151	182	191	117	120	105	118	58	100	91	68
	ALP	50	50	59	52	70	58	63	51	60	55	69	65	67
	T.PROTEIN	5.3	5.5	5.2	5.3	5.2	5.3	5.2	5.2	5.0	4.9	5.2	5.4	5.5
	S.ALBUMIN	2.7	2.4	2.6	2.4	2.8	2.7	2.5	2.7	2.7	2.4	2.4	2.5	2.8
	INR	4.9	2.2	3.7	3.8	1.7	1.6	2.2	1.9	1.7	1.7	2.2	2.2	2.4
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 3 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	52	52	52	52	52	52	52	52	52	52	52	52	52
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	76000	81000	83000	88000	80000	73000	74000	90000	80000	75000	81000	84000	87000
	HB	5.8	6.3	5.8	6.1	5.9	6.4	5.7	5.6	6.1	5.3	5.4	5	5.2
	PCV	23	20	23	25	19	24	24	20	18	22	20	27	20
	TC (in thousands)	12.1	5.9	6.6	5.0	9.0	7.8	9.8	4.7	4.7	6.1	4.8	6.5	9.4
	UREA	41	32	34	55	40	37	46	53	32	39	36	49	50
	CREATINE	1.2	1.1	1.3	1.1	1.5	1.2	1.4	1.2	1.0	1.1	1.0	1.3	1.4
	SODIUM	131	128	132	127	130	129	136	134	129	134	133	132	137
	POTASSIUM	4.1	3.4	3.6	4.2	2.8	3.4	3.1	3.1	3.6	3.1	4.1	2.6	2.9
	T.BILIRUBIN	11.1	12.4	13.8	11.0	11.1	7.4	6.3	6.7	7.4	2.5	3.6	4.0	3.1
SUBJECT 16	D.BILIRUBIN	3.5	3.3	3.9	3.6	2.2	3.0	3.2	2.6	3.1	1.5	1.1	1.1	2.0
	OT	381	297	202	338	294	187	179	191	192	59	74	73	68
	PT	189	183	153	164	166	110	148	103	101	83	94	59	93
	ALP	55	55	63	60	63	66	58	60	70	55	53	65	68
	T.PROTEIN	5.4	5.0	5.1	5.0	5.2	5.2	5.5	5.4	5.0	5.2	5.0	4.9	5.4
	S.ALBUMIN	2.8	2.5	2.5	2.5	2.5	2.7	2.8	2.7	2.5	2.7	2.5	2.5	2.6
	INR	4	3.0	3.3	1.9	1.9	2.3	2.3	1.5	2.2	1.9	2.2	1.9	1.5
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	62	62	62	62	62	62	62	62	62	62	62	62	62
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	74000	72000	74000	79000	70000	64000	65000	80000	71000	66000	72000	75000	78000
	HB	8.2	8.5	8	8.3	8.1	8.6	7.9	7.8	8.3	7.5	7.6	7.2	7.4
	PCV	25	21	24	19	18	26	24	22	26	20	18	20	18
	TC (in thousands)	12.9	4.0	9.7	9.4	7.7	6.7	7.6	8.8	4.3	5.7	5.6	7.1	8.0
	UREA	44	31	39	39	42	37	43	45	37	47	46	48	30
	CREATINE	0.9	1.1	1.0	1.1	1.1	1.5	1.4	1.2	1.4	1.3	1.3	1.4	1.0
	SODIUM	133	135	128	135	136	134	128	131	137	129	136	137	135
	POTASSIUM	4	3.2	4.8	3.1	4.6	2.4	3.4	3.4	4.2	3.9	3.5	3.9	2.9
	T.BILIRUBIN	12.5	12.7	13.1	15.1	11.9	10.9	6.1	5.7	6.4	2.0	2.3	2.6	1.4
SUBJECT 17	D.BILIRUBIN	3.6	2.9	2.9	3.5	2.4	2.7	3.1	3.1	2.9	1.1	1.0	0.9	1.0
	OT	361	279	497	479	368	194	194	193	199	57	75	68	57
	PT	171	178	171	183	189	118	111	124	127	64	87	85	63
	ALP	82	54	65	70	55	61	61	62	69	65	55	52	68
	T.PROTEIN	5.8	5.4	5.1	5.3	5.5	5.4	4.9	5.6	5.5	5.1	5.5	5.3	4.9
	S.ALBUMIN	2.7	2.7	2.5	2.7	2.4	2.6	2.6	2.5	2.5	2.7	2.6	2.6	2.5
	INR	4.1	3.1	3.3	1.6	2.2	1.6	2.5	2.0	2.0	1.7	2.4	2.1	1.8
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	44	44	44	44	44	44	44	44	44	44	44	44	44
	SEX	F	F	F	F	F	F	F	F	F	F	F	F	F
	PLATELET COUNT	38000	36000	38000	43000	34000	28000	29000	44000	35000	30000	36000	39000	42000
	HB	6.1	5.6	5.1	5.4	5.2	5.7	5	4.9	5.4	4.6	4.7	4.3	4.5

	PCV	24	20	19	27	18	22	18	21	21	18	18	18	26
	TC (in thousands)	7.1	5.9	6.1	9.6	8.9	9.2	4.8	8.2	6.4	4.2	10.3	10.6	8.0
	UREA	54	49	39	48	35	34	52	55	34	50	51	35	43
	CREATINE	1.3	1.4	1.3	1.0	1.4	1.3	1.1	1.2	1.4	1.5	1.1	1.5	1.3
	SODIUM	135	137	132	135	135	131	128	127	135	133	137	137	129
	POTASSIUM	41	4.6	3.0	4.2	2.8	2.9	2.9	2.6	2.4	4.0	3.0	3.3	4.4
	T.BILIRUBIN	13.1	11.5	12.7	15.7	11.7	14.9	6.9	7.9	7.2	3.6	3.4	3.9	2.3
SUBJECT 18	D.BILIRUBIN	5.1	3.4	2.0	3.7	3.4	2.5	2.2	2.9	3.9	1.2	1.4	1.8	0.9
	OT	258	236	402	428	334	175	181	178	175	69	58	64	71
	PT	133	151	199	150	173	147	126	144	132	74	53	70	57
	ALP	53	52	50	57	52	59	53	63	68	51	60	52	64
	T.PROTEIN	5.5	5.4	5.0	5.4	5.4	5.5	4.8	5.5	5.3	5.3	5.3	4.9	5.5
	S.ALBUMIN	2.6	2.5	2.7	2.5	2.7	2.4	2.7	2.6	2.7	2.5	2.5	2.7	2.6
	INR	4.1	2.7	4.2	2.0	2.2	1.9	1.8	2.3	2.3	2.5	1.8	2.1	1.8
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 3 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis
	AGE	47	47	47	47	47	47	47	47	47	47	47	47	47
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	70000	72000	74000	79000	70000	64000	65000	80000	71000	66000	72000	75000	78000
	HB	10.5	10.6	10.1	10.4	10.2	10.7	10	9.9	10.4	9.6	9.7	9.3	9.5
	PCV	30	25	21	27	25	19	24	20	20	20	18	23	26
	TC (in thousands)	7.2	9.2	7.7	6.3	8.8	8.7	5.4	9.2	5.8	9.4	9.4	5.7	8.5
	UREA	35	52	54	55	43	39	42	42	54	45	53	40	44
	CREATINE	1.1	1.5	1.1	1.3	1.3	1.1	1.5	1.3	1.1	1.2	1.1	1.1	1.3
	SODIUM	140	135	135	127	133	129	129	128	137	132	134	131	136
	POTASSIUM	4	3.8	3.0	2.9	3.7	2.6	3.1	2.6	2.9	3.0	4.7	3.6	3.3
	T.BILIRUBIN	10.8	11.2	13.1	13.5	13.9	7.4	5.7	6.6	7.8	3.9	3.5	3.2	3.3
SUBJECT 19	D.BILIRUBIN	4	2.0	3.0	3.1	3.1	2.1	2.4	3.5	3.8	1.9	0.9	0.9	1.8
	OT	391	423	362	422	205	195	196	193	193	67	70	58	65
	PT	148	161	191	164	175	124	121	127	119	86	55	99	88
	ALP	76	51	70	67	64	55	62	53	65	69	60	51	60
	T.PROTEIN	5.7	5.4	5.3	5.4	5.5	5.4	5.2	5.5	5.2	5.3	4.8	5.4	5.4
	S.ALBUMIN	2.9	2.7	2.5	2.5	2.7	2.5	2.5	2.7	2.5	2.8	2.6	2.5	2.4
	INR	4.2	3.8	3.6	2.1	2.1	1.7	2.1	2.3	2.2	1.7	1.8	1.6	2.2
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	46	46	46	46	46	46	46	46	46	46	46	46	46
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	60000	61000	63000	68000	59000	53000	54000	69000	60000	55000	61000	64000	67000
	HB	10.4	10.5	10	10.3	10.1	10.6	9.9	9.8	10.3	9.5	9.6	9.2	9.4
	PCV	28	25	19	23	22	21	20	26	24	27	19	19	18
	TC (in thousands)	9.7	8.7	6.9	9.4	8.1	8.3	7.6	8.4	10.2	4.6	8.2	7.6	5.5
	UREA	44	43	34	48	43	38	30	31	52	44	34	55	31
	CREATINE	1.3	1.3	1.0	1.1	1.1	1.4	1.2	1.2	1.0	1.5	1.3	1.2	1.0
	SODIUM	128	132	135	134	130	127	134	132	127	134	131	131	136
	POTASSIUM	3.2	3.1	2.5	3.6	2.8	4.6	2.9	4.0	3.8	4.5	4.2	4.4	2.4
	T.BILIRUBIN	11.8	12.8	12.4	12.4	12.4	7.7	7.7	5.2	7.8	3.0	2.1	3.2	3.8
SUBJECT 20	D.BILIRUBIN	5.1	3.4	3.8	2.7	3.5	3.9	3.5	3.4	3.6	1.2	0.7	1.8	1.8
	OT	394	303	200	334	266	182	197	175	182	59	69	50	66
	PT	111	192	157	151	160	102	120	100	101	54	60	78	95
	ALP	48	57	70	50	57	56	57	70	58	59	64	65	65
	T.PROTEIN	5.4	4.9	5.3	5.3	4.9	5.2	5.1	5.3	5.0	4.8	5.2	5.4	5.0
	S.ALBUMIN	2.7	2.6	2.5	2.4	2.8	2.5	2.6	2.6	2.5	2.8	2.6	2.5	2.8
	INR	5.3	3.4	3.7	1.6	2.5	1.6	1.6	1.6	2.3	1.7	1.8	1.6	2.5
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 3 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	52	52	52	52	52	52	52	52	52	52	52	52	52

	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	29000	28000	30000	35000	26000	20000	21000	36000	27000	22000	28000	31000	34000
	HB	10	9.3	8.8	9.1	8.9	9.4	8.7	8.6	9.1	8.3	8.4	8	8.2
	PCV	29	21	25	27	18	18	23	20	18	27	21	19	19
	TC (in thousands)	7.5	6.4	5.4	7.1	5.6	8.4	5.4	7.9	4.9	5.0	7.3	6.7	8.2
	UREA	34	32	35	47	32	44	31	49	40	34	47	32	33
	CREATINE	1.1	1.2	1.1	1.0	1.4	1.1	1.1	1.1	1.1	1.0	1.3	1.5	1.4
	SODIUM	141	136	135	136	129	131	131	135	137	135	129	132	130
	POTASSIUM	3.4	4.5	4.2	4.4	3.8	2.6	3.3	3.8	2.7	2.7	3.2	2.6	2.9
	T.BILIRUBIN	11.7	13.0	12.2	12.9	13.9	5.6	6.8	6.7	6.5	3.9	2.3	2.2	2.7
SUBJECT 21	D.BILIRUBIN	4.8	2.1	2.1	3.8	3.4	2.5	2.7	3.9	3.4	1.4	0.6	0.9	1.6
	OT	445	259	211	355	217	180	187	190	187	57	69	56	66
	PT	122	155	200	192	177	103	114	108	110	78	57	60	67
	ALP	84	70	60	68	69	60	60	69	56	65	60	69	59
	T.PROTEIN	5.4	5.5	5.1	5.0	5.6	5.3	5.1	5.4	5.6	5.3	5.0	5.4	5.1
	S.ALBUMIN	2.4	2.4	2.7	2.7	2.4	2.5	2.7	2.5	2.7	2.8	2.5	2.5	2.5
	INR	4.1	3.2	3.5	3.9	2.1	1.7	1.8	2.4	1.9	2.3	1.8	1.6	2.0
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 3 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	51	51	51	51	51	51	51	51	51	51	51	51	51
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	78000	78000	80000	85000	76000	70000	71000	86000	77000	72000	78000	8100	84000
	HB	7.2	6.8	6.3	6.6	6.4	6.9	6.2	6.1	6.6	5.8	5.9	5.5	5.7
	PCV	24	23	23	25	25	24	22	27	26	24	26	20	20
	TC (in thousands)	14.3	10.7	4.6	7.1	6.1	4.8	5.6	9.2	11.0	8.3	6.1	6.0	10.8
	UREA	42	33	30	36	49	53	37	34	33	39	43	54	39
	CREATINE	1.5	1.0	1.2	1.4	1.4	1.3	1.3	1.1	1.1	1.2	1.2	1.3	1.1
	SODIUM	133	132	132	137	131	131	131	131	134	134	133	132	132
	POTASSIUM	4.1	3.2	4.5	4.8	3.0	3.5	3.3	3.0	3.2	4.8	3.3	7.4	4.7
	T.BILIRUBIN	14.4	11.6	12.9	13.6	13.6	5.2	7.5	6.8	5.5	6.5	3.4	6.8	5.1
SUBJECT 22	D.BILIRUBIN	5.9	2.3	3.8	3.9	3.9	3.3	3.2	3.1	3.3	3.9	1.6	2.5	3.8
	OT	419	339	204	383	207	177	178	175	194	74	260	156	51
	PT	251	171	155	188	187	143	141	126	134	64	96	88	69
	ALP	46	60	55	60	67	64	51	62	50	63	70	64	61
	T.PROTEIN	5.1	4.8	5.5	5.6	5.5	5.2	5.4	4.9	5.3	5.3	4.9	5.2	5.3
	S.ALBUMIN	2.4	2.7	2.6	2.5	2.7	2.7	2.8	2.8	2.5	2.5	2.5	2.4	2.5
	INR	4.4	3.7	3.2	2.0	2.5	1.8	1.6	2.2	2.2	1.6	3.4	2.0	2.3
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	62	62	62	62	62	62	62	62	62	62	62	62	62
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	75000	74000	76000	81000	72000	66000	67000	82000	73000	68000	74000	77000	80000
	HB	9.9	10	9.5	9.8	9.6	10.1	9.4	9.3	9.8	9	9.1	8.7	8.9
	PCV	34	28	28	26	31	32	25	31	28	30	26	26	25
	TC (in thousands)	15	6.9	9.3	5.1	8.8	5.9	9.9	4.7	10.6	10.7	8.8	4.5	6.4
	UREA	43	33	32	43	34	32	44	41	49	54	55	37	33
	CREATINE	1	1.2	1.4	1.4	1.4	1.0	1.5	1.4	1.1	1.3	1.1	1.3	1.3
	SODIUM	125	136	137	129	128	131	131	130	129	134	130	130	130
	POTASSIUM	4.1	3.3	3.9	4.2	4.3	3.4	3.6	4.0	4.2	3.3	2.8	4.3	2.5
	T.BILIRUBIN	12.9	13.4	11.2	11.6	11.7	7.6	7.0	6.7	5.1	3.0	2.7	3.6	2.2
SUBJECT 23	D.BILIRUBIN	3.9	3.0	3.5	3.2	2.3	3.7	2.4	2.6	3.1	1.9	0.8	0.6	1.2
	OT	425	488	439	219	254	175	197	197	188	51	51	69	66
	PT	231	187	171	189	181	138	106	102	114	74	69	45	47
	ALP	72	67	62	65	52	67	68	69	69	62	63	67	57
	T.PROTEIN	5.2	5.0	5.0	5.0	5.1	5.0	4.9	5.3	5.3	5.2	5.0	5.0	5.5
	S.ALBUMIN	2.6	2.7	2.5	2.7	2.5	2.5	2.5	2.7	2.7	2.8	2.5	2.4	2.6
	INR	4.6	3.2	3.7	1.8	2.5	1.8	1.7	2.3	2.2	1.6	1.7	2.1	2.3
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD

	UPPER GI ENDOSCOPY	GRADE 2 EV											GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	59	59	59	59	59	59	59	59	59	59	59	59	59
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	30000	30000	32000	37000	28000	22000	23000	38000	29999	24000	30000	33000	36000
	HB	6.5	6.6	6.1	6.4	6.2	6.7	6	5.9	6.4	5.6	5.7	5.3	5.5
	PCV	21	20	26	25	23	24	19	24	19	20	20	20	20
	TC (in thousands)	12.1	10.3	4.7	8.9	10.0	8.5	10.4	4.8	10.9	5.8	8.5	6.5	7.8
	UREA	47	55	34	42	43	45	38	45	30	34	39	33	33
	CREATINE	1.3	1.2	1.2	1.2	1.1	1.1	1.3	1.3	1.3	1.0	1.2	1.1	1.4
	SODIUM	126	135	129	129	132	133	135	127	127	129	129	136	129
	POTASSIUM	2.4	4.4	3.6	3.3	3.8	2.7	2.6	4.5	2.5	3.4	3.1	3.4	3.6
	T.BILIRUBIN	18.1	11.5	13.3	14.0	13.9	14.5	5.7	5.3	6.6	3.4	2.1	3.0	2.7
SUBJECT 24	D.BILIRUBIN	7.7	3.7	2.7	3.7	3.3	5.3	3.2	2.8	3.0	2.3	1.0	1.1	1.4
	OT	404	343	237	202	490	185	180	190	194	68	58	70	71
	PT	145	183	155	182	151	103	125	121	137	68	90	86	91
	ALP	48	68	51	69	65	54	57	63	68	51	68	66	66
	T.PROTEIN	5.2	4.9	5.1	5.0	5.0	5.0	5.1	5.6	5.2	5.0	4.9	5.5	5.1
	S.ALBUMIN	2.5	2.4	2.8	2.8	2.6	2.8	2.5	2.5	2.8	2.6	2.4	2.6	2.7
	INR	4.4	4.0	3.7	1.9	2.2	3.9	2.1	2.3	2.1	1.6	1.5	1.6	1.7
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 3 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis
	AGE	62	62	62	62	62	62	62	62	62	62	62	62	62
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	98000	92000	94000	99000	90000	84000	85000	100000	91000	86000	92000	95000	98000
	HB	7.1	6.9	6.4	6.7	6.5	7	6.3	6.2	6.7	5.9	6	5.6	5.8
	PCV	24	23	18	26	26	25	18	20	24	21	19	27	25
	TC (in thousands)	15.4	10.4	10.6	7.9	8.0	7.9	9.2	7.7	7.4	10.0	7.1	8.1	10.9
	UREA	61	49	51	46	48	45	47	44	46	41	50	49	53
	CREATINE	0.9	1.2	1.2	1.0	1.3	1.5	1.2	1.1	1.0	1.5	1.4	1.0	1.2
	SODIUM	146	133	131	128	127	135	136	132	135	135	136	130	131
	POTASSIUM	3.1	2.6	4.4	2.7	3.0	3.1	4.1	2.7	3.8	3.0	3.2	4.7	2.4
	T.BILIRUBIN	10.1	13.8	14.8	12.7	13.1	7.4	6.1	5.1	7.8	2.7	3.0	3.2	3.5
SUBJECT 25	D.BILIRUBIN	4.4	3.9	3.1	2.5	3.1	3.0	2.7	3.6	2.6	1.8	2.3	2.2	2.2
	OT	409	458	312	403	332	196	188	194	194	70	52	63	95
	PT	134	177	191	161	151	140	110	133	105	62	81	54	85
	ALP	67	50	59	56	55	63	64	53	53	53	53	66	58
	T.PROTEIN	5.7	4.9	5.0	5.6	4.8	5.3	5.1	5.2	5.0	4.8	4.9	5.2	5.2
	S.ALBUMIN	2.6	2.4	2.7	2.7	2.4	2.5	2.5	2.6	2.7	2.4	2.4	2.5	2.8
	INR	3.5	4.1	3.4	2.9	1.8	1.9	1.7	2.1	1.9	1.6	1.6	2.0	2.2
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 3 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	45	45	45	45	45	45	45	45	45	45	45	45	45
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	80000	80000	82000	97000	78000	72000	73000	88600	79600	74600	80600	83600	86000
	HB	8.2	8.1	7.6	7.9	7.7	8.2	7.5	7.4	7.9	7.1	7.2	6.8	7
	PCV	25	18	19	27	26	18	19	25	18	23	19	23	20
	TC (in thousands)	11.8	9.5	10.5	4.8	5.6	10.0	5.9	7.0	7.2	8.4	5.4	9.7	7.2
	UREA	46	48	46	49	45	39	46	44	36	55	39	32	39
	CREATINE	0.8	1.2	1.2	1.3	1.1	1.1	1.1	1.1	1.0	1.2	1.4	1.4	1.2
	SODIUM	143	128	136	128	133	136	129	136	130	128	134	127	130
	POTASSIUM	4.4	2.5	3.4	4.5	4.3	3.8	3.4	3.8	4.5	4.2	3.6	2.7	4.1
	T.BILIRUBIN	12.9	13.0	12.6	13.1	11.3	16.1	5.6	6.3	6.2	3.6	4.4	3.1	3.1
SUBJECT 26	D.BILIRUBIN	4.9	3.1	2.6	2.6	3.6	5.5	3.6	3.9	2.3	2.2	3.3	2.0	1.6
	OT	389	444	392	366	455	178	182	176	181	63	71	68	53
	PT	126	191	164	177	192	128	113	150	150	96	82	96	58
	ALP	57	69	60	61	59	58	58	58	62	53	55	66	50
	T.PROTEIN	5.5	5.3	4.8	5.1	5.2	5.0	5.1	5.5	5.0	5.4	5.0	5.2	5.3
	S.ALBUMIN	2.8	2.5	2.4	2.8	2.4	2.8	2.6	2.6	2.4	2.8	2.7	2.7	2.7
	INR	3.7	4.0	3.1	1.6	4.4	1.8	2.1	2.5	1.7	2.4	1.6	2.3	1.5

	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	63	63	63	63	63	63	63	63	63	63	63	63	63
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	46000	44000	46000	51000	42000	36000	37000	52000	43000	38000	44000	47000	50000
	HB	9.2	9.2	8.7	9	8.8	9.3	8.6	8.5	9	8.2	8.3	7.9	8.1
	PCV	26	18	21	23	23	23	27	23	19	18	22	18	24
	TC (in thousands)	18.9	7.9	8.4	9.4	9.7	8.4	5.7	6.4	8.1	10.4	8.9	5.6	9.5
	UREA	62	36	30	32	48	32	50	35	54	43	47	44	44
	CREATINE	1.3	1.1	1.4	1.4	1.1	1.5	1.3	1.5	1.4	1.3	1.1	1.3	1.1
	SODIUM	126	133	132	134	127	135	134	131	136	133	129	136	133
	POTASSIUM	4.1	4.5	2.5	4.1	3.1	3.3	4.6	2.6	3.1	4.0	4.1	3.5	2.8
	T.BILIRUBIN	10.1	11.5	13.7	13.3	12.6	14.4	5.1	7.8	6.1	4.5	2.8	3.9	3.7
SUBJECT 27	D.BILIRUBIN	2.1	2.5	2.3	2.2	2.6	3.8	2.8	3.3	3.5	2.8	2.1	2.7	3.2
	OT	369	254	304	442	414	182	179	180	189	54	57	71	58
	PT	138	159	188	170	190	131	111	135	115	72	22	54	37
	ALP	71	65	62	58	61	61	62	62	65	69	54	65	55
	T.PROTEIN	5.6	5.4	4.9	5.0	4.8	4.8	5.4	5.2	5.2	5.4	5.6	5.3	4.9
	S.ALBUMIN	2.6	2.6	2.7	2.5	2.4	2.4	2.7	2.7	2.4	2.6	2.5	2.8	2.6
	INR	3.9	3.8	3.2	2.3	2.9	1.6	2.3	1.7	2.4	1.8	2.5	2.2	1.7
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	58	58	58	58	58	58	58	58	58	58	58	58	58
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	101000	99000	101000	106000	97000	91000	92000	107000	98000	93000	99000	102000	105000
	HB	9.3	8.9	8.4	8.7	8.5	9	8.3	8.2	8.7	7.9	8	7.6	7.8
	PCV	32	28	21	26	24	20	23	22	18	27	22	23	25
	TC (in thousands)	5.4	4.4	9.7	8.2	7.7	8.0	5.6	9.7	8.4	5.8	5.6	9.4	5.9
	UREA	36	47	51	55	36	52	39	52	51	32	39	47	54
	CREATINE	0.8	1.3	1.2	1.2	1.3	1.2	1.1	1.3	1.1	1.2	1.2	1.1	1.3
	SODIUM	140	133	137	130	130	130	127	135	136	130	130	137	127
	POTASSIUM	4	4.3	2.6	3.1	3.3	4.7	4.3	4.2	3.0	4.4	4.4	2.5	3.3
	T.BILIRUBIN	10.5	13.1	12.3	13.3	13.0	6.4	6.2	5.8	8.0	3.3	2.0	2.8	7.1
SUBJECT 28	D.BILIRUBIN	4.2	2.8	3.6	3.2	2.7	3.6	2.7	4.0	2.6	1.8	0.8	1.7	3.2
	OT	412	385	426	311	332	185	178	199	175	63	60	244	67
	PT	156	175	171	163	189	100	150	121	147	52	95	111	66
	ALP	40	50	52	61	60	55	65	57	53	66	69	65	67
	T.PROTEIN	5.8	5.3	5.1	5.0	5.0	4.9	4.8	5.2	5.1	5.1	5.3	5.2	5.0
	S.ALBUMIN	2.5	2.5	2.4	2.5	2.7	2.5	2.6	2.4	2.7	2.6	2.5	2.5	2.7
	INR	3.6	3.4	3.7	1.9	2.2	2.3	1.8	1.9	2.2	1.6	1.7	2.8	3.4
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 1 EV												GRADE 1 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	53	53	53	53	53	53	53	53	53	53	53	53	53
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	88000	85000	87000	92000	83000	77000	78000	93000	84000	79000	85000	88000	91000
	HB	8.4	8.2	7.7	8	7.8	8.3	7.6	7.5	8	7.2	7.3	6.9	7.1
	PCV	26	24	22	27	24	26	25	21	26	27	22	22	24
	TC (in thousands)	11.5	9.5	4.2	7.9	8.1	10.6	8.4	11.0	5.1	10.1	7.7	10.9	10.9
	UREA	55	45	52	30	38	50	47	38	51	33	33	51	38
	CREATINE	0.9	1.3	1.3	1.0	1.4	1.4	1.2	1.5	1.4	1.4	1.0	1.0	1.4
	SODIUM	144	131	132	127	132	134	135	137	133	136	133	132	128
	POTASSIUM	4.6	4.8	3.2	4.7	2.8	2.6	3.4	3.2	4.7	3.9	2.7	2.4	3.4
	T.BILIRUBIN	12.6	11.5	13.9	17.1	14.0	11.1	6.7	7.9	6.1	2.5	2.6	2.0	2.2
SUBJECT 29	D.BILIRUBIN	3.8	3.4	2.4	3.0	2.8	4.2	3.0	3.3	2.2	1.1	1.0	1.0	0.4
	OT	385	330	435	307	326	180	195	190	200	64	57	73	57
	PT	142	184	200	163	177	102	129	125	148	83	53	51	60
	ALP	52	50	50	65	69	67	51	66	50	66	61	51	59

	T.PROTEIN	5.5	5.3	5.0	5.1	5.6	5.4	5.2	5.4	4.8	5.5	4.9	5.1	4.9
	S.ALBUMIN	2.6	2.6	2.6	2.7	2.5	2.6	2.6	2.7	2.7	2.8	2.5	2.6	2.8
	INR	3.6	3.1	3.3	1.9	1.6	2.0	1.7	1.5	1.8	2.2	2.3	2.5	2.0
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	61	61	61	61	61	61	61	61	61	61	61	61	61
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	66000	60000	62000	67000	58000	52000	53000	68000	59000	54000	60000	63000	66000
	HB	7.3	7	6.5	6.8	6.6	7.1	6.4	6.3	6.8	6	6.1	5.7	5.9
	PCV	25	24	26	25	26	26	26	20	20	25	27	22	24
	TC (in thousands)	13.6	7.4	9.1	7.1	10.1	6.6	6.3	6.9	10.4	5.4	10.2	6.2	4.9
	UREA	48	33	52	52	32	43	36	36	50	48	55	52	37
	CREATINE	0.7	1.0	1.4	1.2	1.3	1.2	1.4	1.3	1.1	1.0	1.1	1.2	1.1
	SODIUM	139	135	129	132	132	132	134	137	134	134	135	135	135
	POTASSIUM	4.1	3.1	4.0	2.6	2.8	4.4	3.9	4.5	3.3	2.8	3.6	4.8	4.1
	T.BILIRUBIN	12.7	13.7	11.7	12.1	13.1	16.6	5.5	7.9	7.9	2.7	2.1	2.8	2.3
SUBJECT 30	D.BILIRUBIN	3.8	3.0	2.7	2.2	3.5	2.8	3.4	2.1	3.1	2.5	2.1	2.4	2.1
	OT	246	201	362	380	468	187	188	188	199	61	64	69	73
	PT	104	197	180	187	190	134	101	107	133	62	59	54	58
	ALP	73	67	50	67	61	50	53	68	53	62	67	70	65
	T.PROTEIN	5.9	5.3	4.9	5.2	5.6	5.3	5.6	5.4	5.1	5.5	5.3	5.1	5.2
	S.ALBUMIN	2.7	2.6	2.5	2.7	2.5	2.5	2.7	2.7	2.7	2.6	2.5	2.4	2.7
	INR	3.6	3.4	3.7	3.3	3.7	2.1	1.6	2.3	2.4	1.7	2.1	1.6	1.5
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	60	60	60	60	60	60	60	60	60	60	60	60	60
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	68000	67000	64000	66000	69000	58000	55000	62000	66000	58000	59000	51000	57000
	HB	6.6	6.3	5.8	6.1	5.9	6.4	5.7	5.6	6.1	5.3	5.4	5	5.2
	PCV	21	20	18	19	21	25	27	25	18	18	23	24	18
	TC (in thousands)	14.8	7.8	4.9	7.2	10.2	4.3	8.6	4.6	7.0	7.8	10.2	7.0	8.4
	UREA	58	50	50	43	51	33	30	32	52	43	30	49	35
	CREATINE	1.1	1.3	1.4	1.3	1.0	1.2	1.5	1.3	1.3	1.4	1.4	1.2	1.2
	SODIUM	133	127	129	127	135	130	137	129	133	129	133	127	137
	POTASSIUM	4.2	4.5	3.4	4.6	3.2	3.9	4.7	3.5	3.0	2.6	3.4	2.9	3.6
	T.BILIRUBIN	13.6	11.4	12.4	13.8	11.3	14.1	5.6	5.8	7.5	3.5	2.3	3.7	2.9
SUBJECT 31	D.BILIRUBIN	4.5	4.0	3.7	3.0	2.9	3.4	2.4	3.6	3.9	1.4	1.6	1.4	2.1
	OT	377	226	278	258	354	183	192	187	186	50	68	51	60
	PT	109	155	185	188	168	113	107	133	123	52	45	42	55
	ALP	58	56	57	63	67	57	53	51	54	63	51	52	53
	T.PROTEIN	5.8	5.6	5.2	5.4	4.8	5.4	4.9	5.1	5.6	5.4	5.2	4.8	5.3
	S.ALBUMIN	2.6	2.4	2.4	2.5	2.5	2.7	2.8	2.4	2.4	2.5	2.4	2.5	2.4
	INR	3.6	3.2	3.7	2.1	1.9	2.3	2.0	1.9	2.3	2.3	1.7	1.6	2.3
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	49	49	49	49	49	49	49	49	49	49	49	49	49
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	88000	82000	84000	89000	80000	74000	75000	90000	81000	76000	82000	85000	88000
	HB	7.9	7.7	7.2	7.5	7.3	7.8	7.1	7	7.5	6.7	6.8	6.4	6.6
	PCV	23	24	22	19	24	25	27	27	23	24	25	18	24
	TC (in thousands)	9.9	6.8	5.7	5.3	4.4	7.0	6.0	5.5	5.1	4.8	9.2	8.0	5.9
	UREA	56	45	46	45	46	55	49	48	36	45	36	53	36
	CREATINE	1.4	1.0	1.3	1.0	1.2	1.2	1.5	1.0	1.1	1.0	1.5	1.4	1.3
	SODIUM	138	128	135	129	137	131	135	127	135	130	133	137	128
	POTASSIUM	2.8	3.5	2.6	3.8	3.1	2.6	3.9	2.7	2.8	3.2	3.8	2.6	4.0
	T.BILIRUBIN	12.6	13.3	13.0	12.2	12.5	12.9	8.8	6.1	6.9	3.7	3.0	3.6	2.1
SUBJECT 32	D.BILIRUBIN	3.7	2.7	4.0	3.8	2.2	2.9	3.2	2.5	3.4	1.7	2.3	2.7	1.1

	OT	379	353	248	272	327	196	196	177	195	67	65	61	74
	PT	201	193	191	167	183	135	139	132	102	67	64	60	50
	ALP	53	70	61	61	63	57	62	60	50	61	67	64	54
	T.PROTEIN	5.5	5.4	4.9	4.9	5.3	5.2	5.4	5.2	5.1	5.3	4.9	5.1	5.0
	S.ALBUMIN	2.6	2.5	2.6	2.7	2.7	2.4	2.8	2.7	2.5	2.8	2.6	2.7	2.6
	INR	3.1	4.0	3.9	1.7	1.8	2.3	1.6	1.9	2.0	2.5	2.4	2.1	2.1
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	57	57	57	57	57	57	57	57	57	57	57	57	57
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	63000	62000	64000	69000	60000	54000	55000	70000	61000	56000	62000	65000	68000
	HB	7.1	6.5	6	6.3	6.1	6.6	5.9	5.8	6.3	5.5	5.6	5.2	5.4
	PCV	25	24	22	18	23	21	25	25	20	18	27	25	27
	TC (in thousands)	11.4	5.1	9.7	9.1	5.1	6.1	8.3	10.5	7.1	10.1	4.1	4.4	5.1
	UREA	44	33	43	34	38	55	43	45	42	45	38	30	34
	CREATINE	1.2	1.1	1.2	1.2	1.2	1.2	1.1	1.1	1.2	1.2	1.2	1.1	1.0
	SODIUM	126	129	127	136	137	131	135	133	129	130	131	136	136
	POTASSIUM	3.4	3.3	3.0	3.9	4.2	3.8	4.3	3.6	3.1	4.0	2.6	3.5	4.6
	T.BILIRUBIN	10.2	11.0	12.6	11.5	12.2	13.1	13.3	8.9	7.3	3.9	3.0	2.1	2.3
SUBJECT 33	D.BILIRUBIN	2.1	3.0	3.4	3.8	3.8	3.9	2.6	3.2	4.0	2.8	2.1	1.1	1.4
	OT	321	380	285	408	407	375	190	177	185	75	74	99	84
	PT	100	180	160	184	175	122	133	130	141	85	54	89	74
	ALP	64	62	63	52	66	64	70	51	50	70	56	64	66
	T.PROTEIN	5.8	5.1	4.9	5.2	5.3	5.2	5.1	5.1	5.5	4.9	4.8	5.0	5.0
	S.ALBUMIN	2.9	2.7	2.5	2.7	2.6	2.6	2.6	2.8	2.6	2.8	2.5	2.5	2.5
	INR	4.1	3.8	4.2	2.5	2.3	2.2	2.4	2.0	2.3	1.8	1.8	2.0	1.5
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	39	39	39	39	39	39	39	39	39	39	39	39	39
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	46000	47000	48000	54000	46000	59000	55000	54000	58000	51000	43000	50000	51000
	HB	9.1	10	9.5	9.8	9.6	10.1	9.4	9.3	9.8	9	9.1	8.7	8.9
	PCV	28	22	26	18	20	26	22	24	21	22	26	24	20
	TC (in thousands)	17.1	10.8	9.6	6.6	4.0	6.4	6.3	4.4	4.0	11.0	6.2	4.2	5.7
	UREA	54	41	55	38	52	32	38	48	53	32	42	43	46
	CREATINE	1.2	1.2	1.3	1.3	1.2	1.4	1.1	1.1	1.3	1.2	1.1	1.4	1.3
	SODIUM	131	128	137	128	127	127	132	136	137	130	134	132	137
	POTASSIUM	4.6	4.8	3.8	3.0	2.9	3.4	4.1	4.8	4.2	2.6	3.6	3.0	2.6
	T.BILIRUBIN	13.1	13.8	16.7	12.2	14.4	10.5	7.7	8.0	6.0	2.9	4.3	4.0	5.4
SUBJECT 34	D.BILIRUBIN	2.9	3.3	4.9	2.4	3.5	5.1	2.1	3.5	3.0	2.8	3.0	3.2	3.4
	OT	444	500	240	400	284	179	188	194	178	71	55	66	68
	PT	195	161	179	161	183	142	124	107	140	100	72	79	64
	ALP	56	58	65	65	57	60	60	60	53	50	56	51	57
	T.PROTEIN	5.4	5.0	5.2	5.4	5.3	5.0	4.8	5.5	4.9	5.3	5.1	5.4	5.0
	S.ALBUMIN	2.5	2.8	2.6	2.7	2.8	2.7	2.7	2.6	2.8	2.7	2.6	2.5	2.8
	INR	4.7	3.8	3.3	2.4	2.2	1.9	1.7	1.8	2.0	2.0	1.7	1.7	2.0
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 3 EV												GRADE 3 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	50	50	50	50	50	50	50	50	50	50	50	50	50
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	103000	104000	102000	107000	98000	92000	93000	108000	99000	94000	100000	103000	106000
	HB	9.4	9.2	8.7	9	8.8	9.3	8.6	8.5	9	8.2	8.3	7.9	8.1
	PCV	26	24	21	19	24	22	21	19	23	23	23	20	19
	TC (in thousands)	8.8	5.9	9.6	8.8	10.7	9.5	5.9	5.0	4.6	9.4	6.4	10.9	10.1
	UREA	44	30	54	35	42	54	31	47	55	43	31	44	43
	CREATINE	1	1.3	1.2	1.1	1.4	1.3	1.2	1.5	1.5	1.3	1.2	1.5	1.2
	SODIUM	140	130	127	128	127	137	131	135	137	130	127	127	136

	POTASSIUM	4.5	3.0	4.5	4.4	4.4	3.4	2.7	3.0	3.4	3.4	3.0	4.8	3.3
	T.BILIRUBIN	10.8	11.4	13.3	14.4	12.1	15.7	11.9	7.8	7.4	3.3	2.1	2.2	2.3
SUBJECT 35	D.BILIRUBIN	3.2	3.3	3.0	4.1	3.8	4.7	3.3	3.8	3.7	2.5	1.1	0.5	0.5
	OT	380	426	431	228	435	181	188	196	193	66	52	150	152
	PT	147	157	157	199	174	145	145	111	150	56	58	62	91
	ALP	66	69	64	70	55	51	64	58	51	68	50	68	53
	T.PROTEIN	5.7	5.1	5.6	5.4	5.2	5.0	5.2	5.4	5.5	5.3	5.3	5.2	4.9
	S.ALBUMIN	2.5	2.4	2.4	2.7	2.7	2.7	2.5	2.7	2.7	2.6	2.7	2.5	2.4
	INR	4	3.8	3.4	1.7	1.6	1.7	1.6	1.5	1.8	2.1	2.4	2.4	2.4
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	45	45	45	45	45	45	45	45	45	45	45	45	45
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	55000	54000	56000	61000	52000	46000	47000	62000	53000	48000	54000	57000	60000
	HB	10.3	10.9	10.4	10.7	10.5	11	10.3	10.2	10.7	9.9	10	9.6	9.8
	PCV	28	26	20	18	18	26	26	19	27	20	27	23	20
	TC (in thousands)	9.5	7.9	9.8	10.6	4.5	9.7	6.0	6.4	4.8	4.4	6.3	7.7	7.7
	UREA	41	35	55	51	48	35	46	32	30	43	50	44	38
	CREATINE	0.8	1.4	1.3	1.1	1.1	1.1	1.3	1.3	1.4	1.1	1.1	1.2	1.2
	SODIUM	143	132	128	133	132	136	131	134	127	130	132	128	128
	POTASSIUM	4.2	2.7	3.5	4.1	4.2	4.6	2.8	4.1	2.5	2.4	3.3	2.4	2.6
	T.BILIRUBIN	10.6	13.6	13.1	11.5	13.7	9.3	7.1	6.9	5.8	3.2	2.7	3.2	2.8
SUBJECT 36	D.BILIRUBIN	3.6	3.3	3.3	2.3	3.0	2.7	2.8	3.9	3.0	2.5	1.9	2.2	1.6
	OT	363	265	293	349	364	196	182	185	177	117	113	74	61
	PT	114	188	165	174	189	108	145	133	115	79	88	68	58
	ALP	64	66	66	65	53	56	66	51	62	62	61	55	60
	T.PROTEIN	5.7	4.9	5.0	5.5	5.1	5.6	5.0	5.3	5.0	5.2	4.9	4.9	5.1
	S.ALBUMIN	2.6	2.4	2.5	2.5	2.7	2.5	2.6	2.6	2.8	2.5	2.8	2.6	2.6
	INR	3.8	3.6	3.9	1.7	2.1	1.5	2.1	1.7	2.1	2.4	2.4	2.4	1.9
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	55	55	55	55	55	55	55	55	55	55	55	55	55
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	77000	74000	73000	75000	63000	68000	67000	74000	62000	66000	66000	65000	64000
	HB	6.1	5.9	5.4	5.7	5.5	6	5.3	5.2	5.7	4.9	5	4.6	4.8
	PCV	25	23	20	18	27	27	20	23	21	24	25	21	18
	TC (in thousands)	9.1	5.2	6.8	6.5	5.5	10.8	5.7	9.1	4.6	8.1	7.3	7.6	4.1
	UREA	67	55	30	36	53	33	36	54	31	48	30	37	49
	CREATINE	1.2	1.0	1.3	1.1	1.3	1.3	1.1	1.1	1.2	1.3	1.2	1.1	1.2
	SODIUM	137	128	134	130	131	132	135	127	129	137	131	134	131
	POTASSIUM	3.2	4.7	4.2	4.5	4.8	2.6	4.4	3.1	3.7	3.9	4.5	3.8	3.6
	T.BILIRUBIN	11.6	13.3	12.8	15.2	16.7	12.8	7.7	7.4	5.3	5.5	5.5	4.2	4.9
SUBJECT 37	D.BILIRUBIN	3.7	3.3	2.2	3.7	3.8	2.1	3.3	2.7	3.2	4.0	3.9	2.6	3.2
	OT	395	212	427	335	424	200	181	185	188	56	63	75	50
	PT	140	185	191	190	198	118	104	126	136	79	56	73	76
	ALP	60	61	50	59	52	57	53	68	57	69	57	50	66
	T.PROTEIN	5.2	4.9	5.0	4.9	5.1	5.6	4.9	4.9	5.0	5.1	5.0	5.4	4.9
	S.ALBUMIN	2.6	2.6	2.4	2.5	2.7	2.6	2.5	2.4	2.6	2.4	2.6	2.7	2.7
	INR	3.6	3.5	3.7	1.7	2.4	1.9	1.9	2.0	2.4	2.2	1.6	2.2	1.6
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	53	53	53	53	53	53	53	53	53	53	53	53	53
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	95000	90000	92000	97000	88000	82000	83000	98000	89000	84000	90000	93000	96000
	HB	6.6	6.3	5.8	6.1	5.9	6.4	5.7	5.6	6.1	5.3	5.4	5	5.2
	PCV	20	23	20	18	23	25	27	27	21	22	22	18	25
	TC (in thousands)	16.4	9.2	6.9	10.2	6.7	6.6	6.7	5.4	5.2	4.5	8.9	10.8	7.0

	UREA	34	31	32	34	39	49	50	53	44	47	33	30	35
	CREATINE	0.8	1.2	1.0	1.1	1.1	1.3	1.0	1.3	1.1	1.2	1.3	1.2	1.3
	SODIUM	142	130	135	131	130	129	135	136	128	135	136	127	134
	POTASSIUM	2.7	2.5	4.0	4.7	3.5	3.7	4.4	4.2	3.5	3.2	2.4	3.9	3.7
	T.BILIRUBIN	14.1	12.0	13.5	11.3	12.7	11.1	8.4	6.3	7.3	5.1	4.5	5.4	4.5
SUBJECT 38	D.BILIRUBIN	4.7	2.4	2.8	2.5	2.5	3.5	2.9	2.3	2.0	3.6	2.7	3.6	3.7
	OT	411	359	310	491	249	192	191	191	185	60	74	83	96
	PT	238	152	198	153	176	140	111	139	130	49	55	66	94
	ALP	63	64	68	60	58	52	58	64	52	61	53	59	68
	T.PROTEIN	5.4	5.1	5.4	5.3	5.4	5.3	5.5	5.0	5.4	5.5	5.5	5.1	5.4
	S.ALBUMIN	2.6	2.6	2.5	2.5	2.6	2.6	2.6	2.4	2.6	2.4	2.4	2.4	2.6
	INR	4.6	4.0	3.9	1.8	2.3	2.0	1.7	2.3	2.2	1.6	2.2	2.0	2.1
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	68	68	68	68	68	68	68	68	68	68	68	68	68
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	55000	51000	53000	58000	49000	43000	44000	59000	50000	45000	51000	54000	57000
	HB	6.2	6.5	6	6.3	6.1	6.6	5.9	5.8	6.3	5.5	5.6	5.2	5.4
	PCV	21	24	22	21	21	18	18	19	26	22	26	22	27
	TC (in thousands)	7.3	5.8	7.5	8.0	4.2	10.0	6.8	5.8	6.6	10.1	5.1	6.6	5.0
	UREA	58	40	44	37	33	49	37	34	47	51	52	40	53
	CREATINE	1.1	1.4	1.4	1.4	1.5	1.2	1.3	1.5	1.4	1.4	1.0	1.5	1.2
	SODIUM	128	131	129	136	136	130	132	137	129	129	137	136	133
	POTASSIUM	3.2	3.5	2.7	4.0	4.2	4.8	3.3	3.3	3.8	2.6	4.7	3.1	3.1
	T.BILIRUBIN	12.9	11.9	12.4	13.1	15.1	11.1	7.2	7.7	7.0	5.0	5.2	5.6	5.1
SUBJECT 39	D.BILIRUBIN	5.3	2.4	3.9	3.0	3.1	2.2	4.0	2.9	3.6	3.9	3.9	3.1	3.0
	OT	304	262	285	471	423	194	199	198	193	68	57	75	62
	PT	191	166	157	188	157	120	135	106	149	41	52	62	82
	ALP	74	51	52	50	58	64	50	61	52	66	56	63	55
	T.PROTEIN	5.6	5.2	5.3	5.1	5.0	5.1	5.3	5.6	5.4	5.4	4.8	5.3	5.1
	S.ALBUMIN	2.6	2.5	2.7	2.4	2.7	2.5	2.8	2.5	2.6	2.6	2.5	2.5	2.4
	INR	3.9	3.2	3.7	2.1	1.5	2.3	1.6	1.7	1.6	1.9	2.2	1.7	2.5
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE	ALCOHOL INTAKE
	AGE	44	44	44	44	44	44	44	44	44	44	44	44	44
	SEX	M	M	M	M	M	M	M	M	M	M	M	M	M
	PLATELET COUNT	61000	58000	60000	62000	61000	61000	58000	52000	57000	58000	61000	64000	63000
	HB	6.9	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8	6.8
	PCV	26	25	22	22	27	26	24	23	18	25	19	21	27
	TC (in thousands)	10.9	7.5	5.6	8.4	8.8	10.2	8.2	10.9	8.0	5.0	6.5	9.7	9.0
	UREA	34	37	37	42	30	32	35	32	41	51	32	33	38
	CREATINE	0.7	1.4	1.4	1.0	1.3	1.4	1.2	1.1	1.2	1.2	1.1	1.3	1.2
	SODIUM	125	137	132	127	137	134	131	130	131	130	129	128	129
	POTASSIUM	4.2	3.3	4.4	3.3	3.2	4.8	3.3	2.7	3.4	2.7	4.3	3.4	3.3
	T.BILIRUBIN	12.1	11.4	12.4	13.4	12.9	10.2	8.8	6.5	6.9	3.7	2.6	3.3	3.5
SUBJECT 40	D.BILIRUBIN	5.1	3.8	2.2	3.7	3.0	2.5	3.2	2.1	3.7	2.0	2.0	2.4	2.3
	OT	450	470	340	383	312	188	175	176	199	142	135	75	82
	PT	155	181	171	176	162	100	107	141	131	83	77	52	63
	ALP	60	55	60	65	69	66	69	60	69	68	66	51	61
	T.PROTEIN	5.4	4.8	5.2	5.4	4.8	5.3	4.9	5.0	5.0	5.3	5.0	5.1	4.8
	S.ALBUMIN	2.5	2.5	2.5	2.5	2.7	2.6	2.7	2.6	2.7	2.4	2.4	2.5	2.7
	INR	4.4	3.9	3.8	1.8	2.2	1.8	2.4	2.1	2.5	1.7	2.0	2.1	2.5
	VIRAL HEPATITIS MARKERS	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE
	GLYCAEMIC PROFILE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL
	ULTRA SOUND ABDOMEN	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD	CLD
	UPPER GI ENDOSCOPY	GRADE 2 EV												GRADE 2 EV
	CAUSE OF HEPATITIS	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis	Portal Vein Thrombosis